The Journal
OF THE KANSAS MEDICAL SOCIETY

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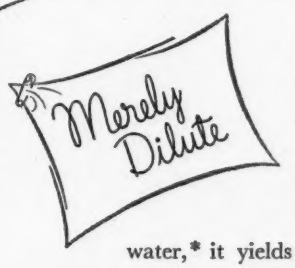

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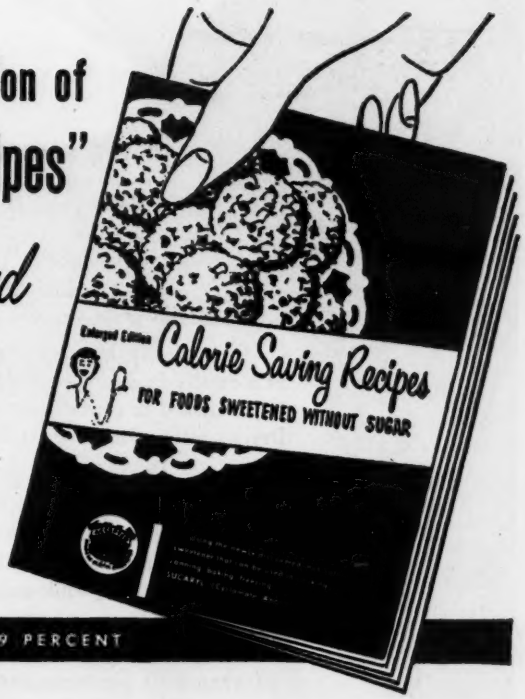

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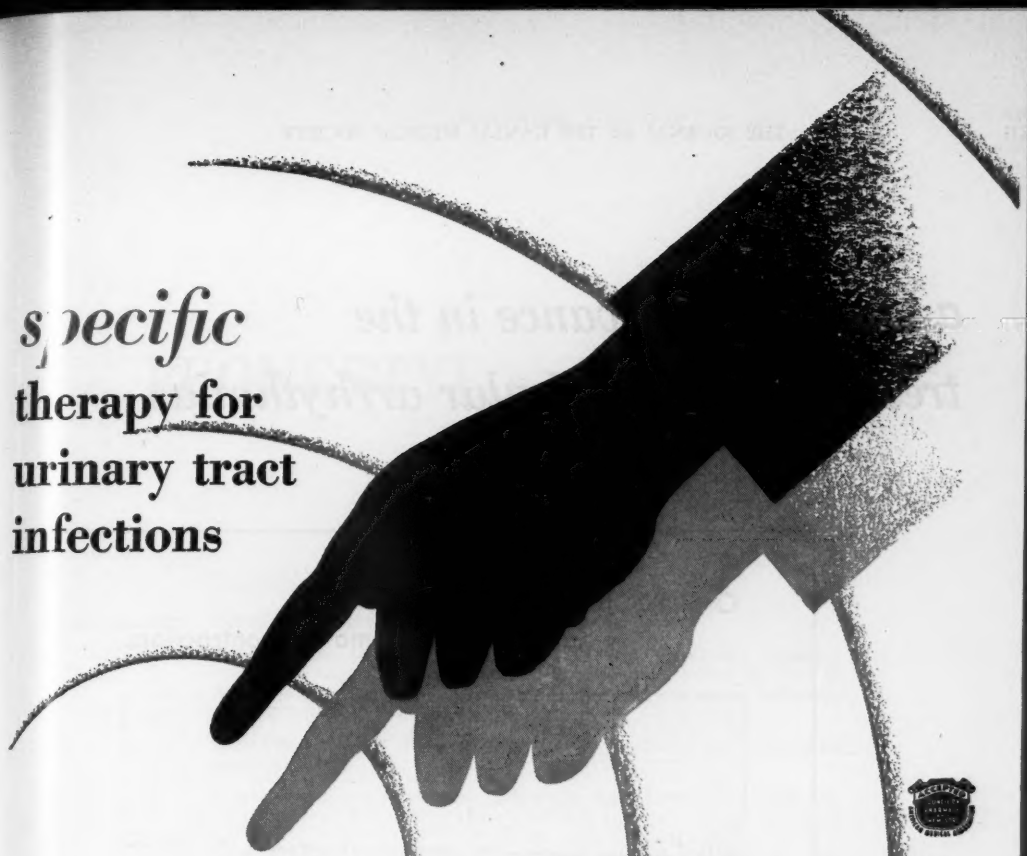

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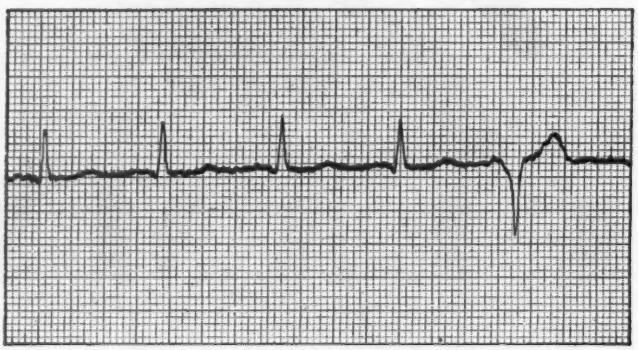
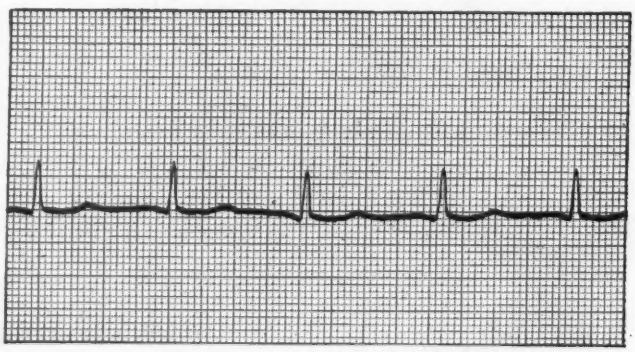


Lead I. Control tracing, ventricular premature contraction.



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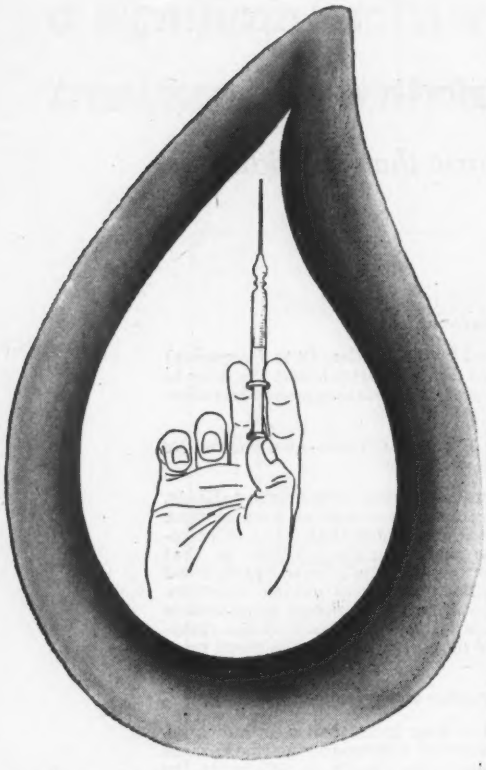

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1. Cannon, P. R.: Recent Advances in Nutrition with Particular Reference to Protein Metabolism, Lawrence, Kansas, University of Kansas Press, 1950, pp. 56-60.

2. Rose, W. C.: The Nutritive Role of the Amino Acids, The Science of Nutrition, New York, The Nutrition Foundation, 1946.

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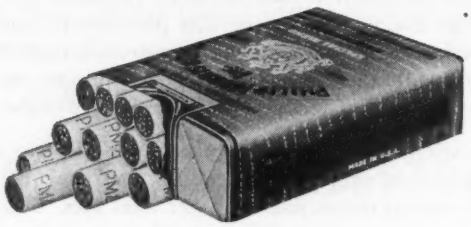

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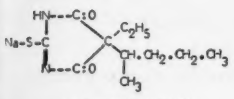
Surital Sodium*†
I. Clinical Use

Paul H. Lorhan, M.D., Gretchen Guernsey, M.D.,
and Marguerite M. Devine, M.D.

Kansas City, Kansas

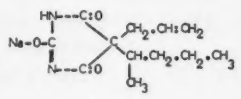
Surital sodium, a thiobarbiturate relatively new to clinical anesthesia, has been used in 200 cases at the University of Kansas Hospital. This paper will give a brief description of the drug, review some reports of its use by others, and present a preliminary evaluation of our experience with it.

Chemically, this agent is sodium 5-allyl-5 (1-methyl)-butyl-2-thiobarbiturate; its relation to allied drugs is apparent from the following structural formulae:



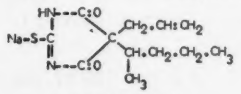
PENTOTHAL SODIUM

(Sodium ethyl (1-methyl) butyl thiobarbiturate)



SECONAL

(Sodium allyl (1-methyl) butyl barbiturate)



SURITAL SODIUM

(Sodium allyl (1-methyl) butyl thiobarbiturate)

The numerous barbiturates in clinical use have similar pharmacological properties; the major variations occur in lag in onset, duration of depressant action, and suitability for use as intravenous agents. In surital sodium we can expect rapid potent action due to the introduction of sulphur and the unsaturation of a side chain.

Surital sodium is supplied in sealed ampoules containing one gram of the dry powder buffered with a small amount of sodium carbonate. Solutions are prepared and used in exactly the same manner as solutions of pentothal sodium. We use a concentration of 2.5 per cent in the majority of cases, and have found distilled water, normal saline, and five per cent glucose equally satisfactory as solvents. The clear, light yellow alkaline solution is quite stable at room temperature, but occasionally a batch becomes cloudy upon standing for a variable period, in which case we discard it. Helrich, Papper and Rovenstine[1] report solutions kept 14 days at room temperature without change in appearance or potency, but they recommend using only normal saline as the solvent. Dornette and Tuohy[2] state that the solution decreases in potency unless kept refrigerated.

Pharmacologically, the major effect of surital sodium is on the nervous system. The central nervous system depression which makes it useful as an anesthetic is also its greatest potential hazard. Intravenous injection of a suitable dose produces loss of consciousness in a few seconds and surgical anesthesia within a few minutes. The "suitable dose," like that for any other agent, is determined by the clinical signs during induction and maintenance. If skillfully administered for short procedures (under 15 minutes), it is possible to have the patient awake within three minutes after the operation is completed. For example: In the case of a 64-year-old, obese woman requiring closed reduction of a wrist fracture, 10 cc. of a 2.5 per cent solution of surital sodium provided satisfactory anesthesia following premedication with only atropine gr. 1/150. The procedure took 10 minutes; at the end the patient was awake, comfortable, and talking rationally.

In using surital sodium, one gets the clinical impression that in many comparable cases there is a lower anesthetic dose and shorter awakening time than is seen with pentothal sodium. Since dosage varies within rather wide limits depending on both

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†The surital sodium used in this study was supplied by Parke, Davis and Company.

patient and anesthesiologist, further study is necessary to confirm or deny this impression.

While it cannot be stated with certainty that surital sodium has important effects on any tissue except the nervous system, there is considerable controversy in the literature regarding the action of all thiobarbiturates on certain other organs, mainly the heart and liver.

A primary cardio-toxic action, postulated by some workers, is not confirmed by the laboratory and clinical experience of others. Wyngaarden et al,[3] investigating this problem at the University of Michigan, report: "In the heart-lung preparation, the thiobarbiturates, pentothal and surital, exhibit only a moderate degree of cardiac toxicity, which is no greater than that produced by the corresponding oxygen analogues. Surital is perhaps less toxic, but definitely not more toxic, than pentothal." However, even if these drugs proved to be absolutely innocuous in their direct action on the cardiovascular system, there remain two mechanisms by which they can be indirectly cardio-toxic: (1) Hypoxia from respiratory depression or obstruction. In clinical practice, this is by far the greatest toxic hazard and the most frequent cause of death during intravenous anesthesia regardless of the agent used. Even moderate hypoxia may be poorly tolerated by an already damaged myocardium: or, if the patient survives, he may have irreversible cortical changes. (2) The parasympathomimetic properties of thiobarbiturates may produce arrhythmias, bradycardia, or hypotension.

Hepatic disease is listed as a contraindication to the use of thiobarbiturates on the theory that these drugs are detoxified in the liver. Actually, knowledge of their metabolism is very incomplete. Animal experiments yield conflicting results, but there is little evidence from clinical reports that thiobarbiturates cause or increase liver damage.

TABLE I

COMPLICATIONS REPORTED IN RECENT LITERATURE

COMPLICATION	HELRICH, PAPPER & ROVENSTINE (1,200 cases)	DILLON & DENSON (700 cases)
laryngospasm	8%; severe in less than 2%	0.34%; total 24 cases severe in 4 cases
bronchospasm	less than 1%	1 case; relieved by change to ether
respiratory depression	15%; severe in 4%	
apnoea		0.38% or 27 cases; duration 2 min. to 30 min. (occurred in 5 cases with pt. moving)
tachypnoea		1 case
Cheyne-Stokes respiration		1 case (89 yr. old male)
cough		10 cases; frequently precedes laryngospasm
hypotension	9%; usually transient but may be significant, esp. in older patients. DUE TO RAPID INJECTION	0.70% or 49 cases (21 cases had B.P. drop of 40 mm. Hg. or more; all of these were 55 yrs. or older
arrhythmias	1 case	2 cases; 1 cleared spontaneously, other with ether
emesis	1 case	3 cases
venous thrombosis	1 case	
asthma	4 pts. with history of asthma given surital; attacks were precipitated in 2 pts.	
other	One death in this series which autopsy showed not related to the anesthetic agent	1 case each; "pentothal shakes," prolonged (24 hr.) sleep, dermal reaction, paralytic ileus
		NO DEATHS
		NO POST-ANESTHETIC COMPLICATIONS

Helrich, Papper and Rovenstine,[1] reporting on the use of surital sodium in 1,200 cases at Bellevue Hospital, conclude that it deserves further trial because in certain respects it may be superior to similar drugs in current popular use. Among these are: (a) More rapid awakening; (b) Less frequent circulatory depression; (c) More rapid restitution of spontaneous breathing after large doses are given rapidly; (d) More benign nature of laryngospasm.

The largest total dose administered by the Bellevue group was 3.5 Gm. Table I shows their complications, as compared with those reported by another group.[4]

In a series of 700 anesthetics administered at the Los Angeles County Hospital, using surital sodium, Dillon and Denson[4] concluded: "The outstanding feature observed was the similarity of action between pentothal and surital . . . It is doubtful if an anesthesiologist using one of the two drugs without knowing which could differentiate between the two solely on the basis of action. The awakening time is somewhat shorter with surital, but is undoubtedly influenced by the skill of the anesthesiologist. The incidence of laryngospasm is the same as that expected with pentothal, but the individual attacks appear more benign; in general, other complications are the same."

Our own experience with surital sodium is limited to 200 cases; this series is too small to justify drawing any general conclusions, and we wish to emphasize that this is merely a preliminary report. However, it is of interest that our complications differ somewhat from those reported by others. Table II shows the complications encountered in all cases in which surital was used; however, when multiple agents are used, it is obviously difficult to locate the offending one.

The relatively high incidence of nausea and emesis contrasts markedly with the reports of other workers; it was not particularly associated with emergency procedures where the patient might have eaten a short time previously; neither was there a high incidence of anoxia in these cases as judged by the usual clinical signs.

An increased number of patients apparently develop a bradycardia with this agent, though in the majority of our cases it neither complicated the procedure nor caused determinable sequellae.

If the nausea, emesis and bradycardia are due to central vagal stimulation, surital appears to be more strongly parasympathomimetic than pentothal; this requires further investigation.

Summary and Conclusions

1. Surital sodium (sodium 5-allyl-5 (1-methyl) butyl thiobarbiturate) is described and some reports of its clinical use are reviewed.

2. A preliminary report of the use of surital sodium as an anesthetic in 200 cases is presented.

TABLE II

COMPLICATIONS OCCURRING WITH SURITAL

Complication	Surital Alone	Surital-Curare	Surital-N_2O-Curare	Surital-N_2O	Surital-Spinal	Surital-Other Gen.
Bradycardia	1		3	2		5
Tachycardia	1		2			2
Arrhythmias		2			1	5
Hypotension	3		3	2		3
Decreased pulse pressure		1				1
Laryngospasm	2	1	1			
Cough	1			1		1
Bronchospasm			1			
Apnoea			1			
Resp. depression						1
Nausea & Emesis	12		12	4	1	1
Headache	2		6	1	2	1
Excitement	1		2		1	1
Urinary Retention			1			2
Nystagmus	1					

3. The clinical manifestations of this agent are very similar to those observed with pentothal sodium; because of some apparent advantages, especially lower dosage and a shorter awakening time, surital sodium deserves further trial.

4. Evidence that surital sodium may be more strongly parasympathomimetic than pentothal sodium has been observed and requires further investigation.

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Surital Sodium*
II. Comparison with Pentothal Sodium

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The statement has been made that surital sodium (sodium 5-allyl-5- (1-methyl) butyl 2-thiobarbiturate) is a more potent hypnotic than pentothal sodium. Kelly et al,[1] in laboratory studies on the dog, found surital to be 139 per cent as potent; a similar study by Wyngaarden et al[2] shows surital to be 150 per cent as potent as pentothal. A clinical survey by Dornette and Tuohy[3] found no significant difference in dosage per minute between the two drugs, although they report slightly smaller doses of surital in comparable cases.

Our impressions after a preliminary trial of surital sodium have been previously described.[4] In order to compare this agent more objectively with pentothal sodium, we analyzed 200 consecutive cases in which the latter was the sole anesthetic agent. Table I shows respective dosages.

These figures indicate that any advantages from the lower dose of surital are gained chiefly in short procedures. We do not advocate the use of unsupplemented thiobarbiturates for long operations; those few cases which lasted an hour or more represent chiefly head and neck surgery which was unexpectedly prolonged. It is justifiable not to interrupt such procedures as long as the patient's condition is satisfactory.

Complications with these two agents are shown in Table II.

It should again be emphasized that the doses and complications reported here are for those cases in

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TABLE I

COMPARATIVE ANALYSIS

ANESTHETIC TIME	PENTOTHAL SODIUM (200 cases)	SURITAL SODIUM (94 cases)
Under 15 min.	77 cases Av. Time 10.6 min/case Av. Dose 452.5 mgm/case Av. Dose 41.7 mgm/min.	47 cases Av. Time 11.4 min/case Av. Dose 395.0 mg/case Av. Dose 34.6 mg/min.
15 to 30 min.	81 cases Av. Time 22.2 min/case Av. Dose 657.1 mgm/case Av. Dose 29.6 mgm/min.	31 cases Av. Time 22.3 min/case Av. Dose 479.0 mgm/case Av. Dose 21.4 mgm/min.
30 to 84 min.	42 cases Av. Time 38.5 min/case Av. Dose 830 mgm/case Av. Dose 21.5 mgm/min.	16 cases Av. Time 40.8 min/case Av. Dose 815.6 mgm/case Av. Dose 19.9 mgm/min.

TABLE II

COMPARATIVE ANALYSIS OF COMPLICATIONS

Type	Pentothal (200 cases)	Surital (103 cases)
Respiratory		
Cyanosis	2	----
Severe resp. depression	1	----
Apnoea	1	----
Sneezing	2	----
Coughing	3	1
Laryngospasm	4	3
Circulatory		
Bradycardia	----	1
Tachycardia	1	1
Arrhythmia	2	----
Hypotension	3	3
Miscellaneous		
N & E	3	12
Headache	1	2
Irrationality	2	1
Nystagmus	----	1

which the thiobarbiturates were the sole anesthetic agents used, and no curarizing agents were given. The majority of patients received premedication with one of the belladonna drugs plus either an opiate or demerol in appropriate doses.

We found no significant difference in either incidence or severity of complications except for the previously noted[4] nausea and emesis with surital.

While the incidence of bradycardia was not significantly increased in those cases where surital was the sole anesthetic agent, the high incidence previously reported[4] still remains to be explained.

Summary

Surital sodium and pentothal sodium are compared as regards dosage and complications.

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The Treatment of an Attack of Bronchial Asthma*

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There are two aspects to the treatment of bronchial asthma; the first, of interest to the general practitioner, the treatment of the wheezing itself; and the second, perhaps less immediate, the general therapy of the asthmatic patient.

The treatment of bronchial asthma has progressed as far in the last 10 years as have the methods of treatment used in surgery, medicine, and their subsidiary fields. A physician who treats acute bronchospasm with an injection of morphine is criminally negligent, and one who limits himself to injecting epinephrine may have given his patient only temporary relief. We now know that he has done his patient a real dis-service unless he follows this with the other medications known to be necessary to prevent an immediate recurrence. For the patient in acute bronchospasm of moderately severe degree, an injection of epinephrine alone is not usually enough.

A patient with bronchial asthma is a sick man suffering from a condition which, if allergic and in United States, causes 7.2 per cent of the total number of illnesses each year, two per cent of the days of disability annually incurred, and 0.2 per cent of the annual deaths. If the vital statistics of hay fever and bronchial asthma are taken, they are found responsible for 3,450 medical cases each year. The total days of disability is estimated at 22,000,000 with a disability rate of 171/1,000, 2,100 annual deaths, and a mortality rate of 1.7 per cent/100,000. A good number of those patients who die, do not expire as a result of the bronchial asthma, but equally often, of the treatment given them. In the light of our present knowledge, the treatment of an asthmatic patient in an acute attack now requires the same thorough understanding of the basic conditions present as is necessary in the treatment of cardiac, nephritic, and other states in which there is both a local condition and its systemic, or general, effects.

A patient with severe wheezing can tax, to the utmost, the physician's knowledge of physiology,

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pathology, pharmacology, and psychiatry, as well as all his other knowledge, culture and background, as well as that special sense for ferreting out important facts which is seen at its highest in the allergist and the detective, and without which both may be wasting their time.

The typical asthmatic attack needs no detailed description. Often forgotten, however, is the fact that between attacks, and much less obvious in the absence of symptoms, the signs of bronchial asthma are nevertheless present. Although the cough and wheeze may be occasional, or absent, the dyspnea is always present either clinically or by laboratory studies. The findings show that the average asthmatic patient always presents a lowered vital capacity which is approximately 60 per cent of the normal. When we go through the so-called static measurement of the vital capacity to the dynamic measurement of volume per unit time, it is found that the asthmatic emphysematous patient falls far short of the normal 75 per cent for the first second, 90 per cent for the second second, and 100 per cent for the third second of expiration.

The history is straightforward. The patient tells the physician of the cough, dyspnea, and the expiratory wheeze. The signs and symptoms are deceptively apparent. The bronchospasm makes itself audible and literally shouts the diagnosis. The distinctive sonorous and sibilant rales and rhonchi, changing in pitch and intensity as the stethoscope is applied over different parts of the lungs, which are obviously held in the position of almost maximal distension, are easily recognized. Clinically, the inspiratory and expiratory velocity rates are slowed, and the amount of residual air is therefore increased. The laboratory studies show that the alveolar and blood oxygen saturation levels are below normal, and at first, the blood carbon dioxide and alveolar carbon dioxide, equally low. The amount of reduced capillary hemoglobin is increased, reaching levels of recognizable cyanosis at the five gm./per cent level. There is an increase of serum in the alveoli, and of mucus into the bronchi.

It is equally obvious that the patient is apprehensive, suffering from the effects of successive "alarm reactions." He is thirsty, and undoubtedly dehydrated. He is often hungry because he cannot stop breathing sufficiently long to swallow. He is tired because of the lack of sleep. The hypoxia and cyanosis, the vascular collapse, are all equally evident. Death, when it does occur, is due to asphyxia. To treat the patient adequately, all of these pathological processes must, when possible, be reversed, although some of the sequelae, especially the emphysema, invariably present in the chronic asthmatic patient, are, in fact, irreversible.

The general practitioner, if he has seen the patient before, knows a great deal about him, or if this is the first visit, can quickly take the basic history. The age at onset is important. The differential diagnosis is helped by asking whether other allergic manifestations, such as hay fever, and less often, atopic eczema, urticaria, or drug sensitivity, are present. A knowledge of familial incidence of allergy will point to the correct causes. A history of previous hospitalizations and the treatment and the results, as well as the facts elicited by previous studies, x-rays and skin tests, all help in the diagnosis. More important, the patient's history of relief with previous medications given, such as epinephrine, aminophylline, ephedrine, antihistaminic agents and hyposensitization, environmental or climatological change, indicate the measures to be taken.

Equally obvious is the patient's condition, as acute or chronic, and if of the former, recurrent, or of the latter, of the severity termed "status asthmaticus."

When visiting a patient in the acute asthmatic state, the factors responsible for his condition may not be immediately apparent, although the physician should question whether the attack was preceded by such non-specific factors as pulmonary irritation, due to the inhaling of irritant fumes; pulmonary infection, fatigue, exertion, or emotional distress. The possibility of reaction to unusual seasonal, environmental, or climatic influences should also be elicited, if possible. The information will also be volunteered that the patient has had an unusual exposure to an allergenic food, inhalent, injectant, or drug, all of which help determine the treatment and the prognosis.

Of greatest importance are the concomitant conditions which may not be immediately recognized. The emphysema, which may be generalized, subcutaneous, or mediastinal, is one such. If other complications are present, they must be treated in their own right, and those which should be in the forefront of the mind, must include the common cold, paranasal sinus disease, bronchitis, pneumonia, pleurisy, bronchiectasis, and less likely to be present, bronchostenosis, segmental atelectasis, and perhaps, with great rarity, obliterative endarteritis of the pulmonary vessels.

Some of the conditions associated with wheezing must be considered. The physician should suspect, and also be able to recognize, wheezing as due directly to industrial, chemical, biological or miscellaneous inhalants; to infections, which in addition to those which may be bacterial may also be mycotic, viral, rickettsial, or parasitic; to neoplasms, primarily or secondarily affecting the lungs; to blood diseases

associated with dyspnea, as anemia and perhaps leukemia; to generalized conditions, as lupus, periarteritis nodosa, Loeffler's syndrome, tropical eosinophilia, cystic disease of the pancreas, and rheumatic fever.

In the lung not only must the conditions listed be considered, but also perhaps others associated with pulmonary fibrosis and the circulatory disturbances, as associated with pulmonary edema, with or without cardiac or renal disease, emboli, or infarcts. A high index of suspicion of the signs and symptoms of other types of respiratory obstruction may save the patient's life. Occasionally, when the patient is seen for prophylactic medication between attacks, it is well to make certain that the symptoms described as a wheeze are not, in reality, noisy respiration due to other causes.

A cough may be due to obstruction or reflex irritation of any part of the respiratory tract, and dyspnea, when present, may also be due to many types of extra-pulmonary obstructions, lesions, or infections, or occasionally due to the hyperventilation syndrome, psychologically induced.

The treatment of an asthmatic attack in children requires different handling than that seen in adults. In the child, the differential diagnosis includes pertussis, which may simulate bronchial asthma, being differentiated, of course, by the absence of any history of allergy, the type of cough and the typical whoop, the high lymphocyte count, and the results of throat cultures. In patients in whom the cough is persistent, and in whom there is a lung infection while the patient fails to gain weight and presents stools which are fatty, foul, homologous and large, the diagnosis of cystic fibrosis of the pancreas must be made. In infants, other conditions seen, although rarely, are tuberculous, tracheal, and bronchial nodes as causing intractable cough and asthmatic breathing by pressure upon the tracheo-bronchial tree, the passive bronchial constriction occasionally simulating bronchial asthma, although marked by inspiratory as well as expiratory wheezing. In children, also, especially those who suffer from prolonged or severe dyspnea, the possibility of a foreign body, of "thymic compression stenosis," however rare, and of vascular anomalies, such as a double aortic arch, must be brought up, if only to be considered and their possibility discarded. A rare patient may present a papilloma of the trachea or larynx or an adenopathy, usually tuberculous, and at the bifurcation of the trachea. Wheezing due to psychological trauma must always be considered in any asthmatic attack in a child in whom other signs and symptoms are absent.

From a clinical point of view, in children, cyanosis with shallow breathing, is the cardinal sign of a serious condition. If the breath sounds are feeble and there are moist rales, the patient is dangerously ill. If, on the other hand, the rales decrease after an attack of coughing, the prognosis is better. Forceful respiration and the absence of cyanosis are the signs of mild or moderate bronchospasm. In children, an attack of asthma may be associated with a rise in temperature, although no infection is present. Children, especially asthmatic infants, are often neither apprehensive nor orthopneic.

An injection of epinephrine (1:1000), no more than 0.2-0.4 cc., is both diagnostic and therapeutic, when given subcutaneously to children at intervals of 15-30 minutes. If the dose is too large, and the child is of an age when he can talk, he not only grows pale but complains of pounding headache. He becomes obviously apprehensive, and presents an accelerated pulse within 8-10 minutes. A second or third injection, if necessary, can be given at intervals of 15-30 minutes, but if epinephrine does not work within 5-10 minutes, further injections will usually not be effective. Larger doses make the patient "epinephrine-fast," not only because the drug is not effective, but because the patient has a condition which epinephrine will not help. The wheezing is due, not to bronchiolar spasm, but to bronchial plugging and an edematous bronchial mucosa.

For such plugging, the patient requires an expectorant, or if he is more seriously ill, an emetic. In infants and young children, the drug of choice is syrup of ipecac and the dose is 3-10 cc. This may be followed by the drinking of lukewarm water, which will initiate the so-called peristaltic movements of the bronchial tree, moving the plugs to the ciliated epithelium, and then to the tracheal bifurcation of the larynx, at which point the cough reflex takes over. This has been described as "tracheal vomiting." Ipecac will help a patient raise mucus, which might otherwise take several days to reach the cough center. In milder cases, potassium iodide in the saturated solution with the dose suitable to the age is the expectorant considered most effective. Adjuvant treatment, if the patient is in a condition to take it, is represented by an enema, if the bowels have not moved, and unmedicated steam inhalation, if such appears necessary.

For more severe or prolonged attacks, the patient should be moved to the hospital where he can be given 300-1000 cc. of 10-15 per cent intravenous glucose and sedative medication, namely 5-10 gr. of acetylsalicylic acid (in patients who are not aspirin sensitive, rare in children), triple bromides, 5-15 gr., phenobarbital, up to ½ gr., or if at bedtime, chloral hydrate (3 gr.) orally, in patients who can

swallow capsules. Most children dislike, and will not take, the liquid.

In the hospital, or if necessary, at home, it is usual to give children a salt-free, high carbohydrate, high protein, liquid diet. Psychotherapy is evidenced by a cheerful and reassuring attitude on the part of the patient's family and physician and is an important part of allaying fear, if present.

In the adult patient, there are four states, or grades, of bronchial asthma. In the early stages of the condition, there is a respiratory oppression, associated with difficulty in deep inspiration, which resembles the substernal distress, although in mild degree, seen in patients with coronary heart disease. Such pain is mild, lasts for hours, does not interfere with activity and is not transmitted. The chest is almost always free of signs at this stage.

The next degree is that in which the patient suffers from what appears to be an allergic tracheitis, with a constant cough and dyspnea. Examination of the lungs frequently discovers a subclinical sibilant and sonorous rales. The wheezing during this stage does not occur in paroxysmal attacks.

As the condition grows more severe, the dyspnea becomes more marked and troublesome, becoming expiratory in type, and the patient presents a typical asthmatic attack of a degree which will often disappear of itself, with cessation of exertion, or when more severe, responds readily to epinephrine by injection or by inhalation, or to ephedrine and other drugs taken orally.

When the patient has been in such severe attacks for a day or two despite medication, he is said to be in "status asthmaticus." When such severe asthma continues over a long period of time, the patient making little or no response to treatment, his condition is labelled as "intractable asthma." Such patients may require treatment with Cortisone and ACTH.

The treatment of adults with bronchial asthma, granted that the other conditions listed as important differential diagnoses have been eliminated, is best perhaps discussed from the point of view of the drugs used and the effects to be expected from each.

In mild cases, and while studies are in progress, potassium iodide as an expectorant, in dosage drops of 15-30, taken three times daily, omitting the medication two days each week (Saturday and Sunday) is often enough, although occasional patients need additional medication.

For simple wheezing following exertion, laughter, overeating, emotional disturbances, or mild response to allergenic foods, inhalants, or fumes, the patient may have on hand any of the ephedrine-phenobarbital-aminophylline mixtures, as tablets or capsules. Such as Luasmin, Tedral, Amodrine, Amisec, Quadronal and others, will often satisfactorily relieve the symptoms. For wheezing during the early-morning hours, an enteric-coated tablet and the capsule may both be given at bedtime. Side reactions, such as palpitations, jitteriness and insomnia sometimes occur requiring additional medication. In males, during the middle-age years, there may be spasm of the prostatic sphincter, and diminution of the urinary stream, as well as actual dysuria. Aminophylline, with Benadryl and racephedrine, as in Hydryllin capsules have been particularly useful in such patients, as have the ephedrine-like synthetic drugs, such as Isuprel and Orthoxine, similarly used.

The "Gay treatment of bronchial asthma" consists of the following prescriptions:

Tincture of digitalis	3.0 cc.
Liquid potassium arsenite	9.0 cc.
Potassium Iodide	12.0 gm.
Sodium phenobarbital	0.75 gm.
Saccharin	0.05 gm.
Amaranth (1% as a dye)	1.2 cc.
Water q.s.ad.	180.0 cc.

This is taken in 4-5 cc. doses four times daily, one-half hour before meals, and at bedtime. Another prescription used consists of the same dose of digitalis, potassium arsenite and potassium iodide, with the phenobarbital omitted, and Allurate elixir and Elixir Lactopepsin substituted in equal quantities to 180 cc., no water being used. The dose is exactly the same. After two months, the lunch hour dose is omitted, and then for the next six or eight months one teaspoonful is taken on arising and retiring, and after one year the dose is one teaspoonful daily at bedtime. In addition, the patients are given a calcium preparation, such as calcium lactate. Some patients take Bile salts and Cascara (Parke, Davis and Company). How much of the success of this treatment is due to the medications and how much is psychotherapy would be extremely difficult to evaluate. Personal use of this mixture in older patients is often followed by occasional immediate relief. The effects are, however, not constant nor continuous.

For one who is more seriously ill, epinephrine (1:1000) may be injected subcutaneously, the dose being 0.2-0.5 cc. or inhalation of the 1:100 solution may be used. Relief is generally noted, subjectively, by the patient in 3-10 minutes; and objectively by the physician in 8-15 minutes. Vaponefrin, Isuprel, and Norisodrine powder are often equally effective. In more seriously ill patients, the 0.5-1.0 cc. of epinephrine (1:1000) can be given intravenously in 1000 cc. of glucose saline. If the patient has had epinephrine with no results for several doses, he is

"epinephrine-fast" and additional doses should not, by any manner, be given him. Larger doses cause the production or release of histamine and may actually contribute to additional bronchospasm, edema and dyspnea. Such epinephrine-fastness, however, may disappear following the use of an antihistaminic agent, as Benadryl or Thephorin given in 25-50 mg. doses intravenously. Under such conditions, however, the use of Aminophylline is preferable.

Aminophylline by mouth is absorbed slowly and the relief of symptoms is short in duration. Given intramuscularly, it has little or no effect in most patients, and besides, is painful. Papers on the use of rectal Aminophylline report excellent results. Suppositories are almost equally effective. Aminophylline used intravenously, however, is the method of choice. A 20 cc. solution (0.25-0.5 gm.) can be injected intravenously at a rate of about 2.0 cc./minute in a mildly asthmatic, or moderately ill patient. This dose can be repeated two or three times in each 24-hour period. In those more severely ill, 0.5 gm. can be added to 1000 cc. of glucose saline. This can be run continuously, with a total of 3000 cc. daily for a week or more, depending upon the severity of the asthma, the second daily liter being glucose and distilled water; the third, either glucose-saline or glucose and distilled water, depending upon the patient's general condition. The effect can often be supplemented by Aminophylline and ephedrine, given orally. Other aspects of intravenous therapy will be discussed below.

The safest sedative medication consists of sodium bromide, or triple bromides, in the 5-15 gr. dose, combined with chloral hydrate in the 5-10 gr. dose, in simple syrup or Iso-elixir. Phenobarbital may be used orally, subcutaneously, or by injection. Demerol is usually safe in doses of 50-100 mg. intramuscularly or subcutaneously, or 50-150 mg. orally. Demerol in the 100 mg. dose and Scopolomine hydrobromide in the 0.3 mg. dose, combined for subcutaneous use in the "wet asthmatic" is a useful combination to remember. (Segal)

With patients more severely ill, gas therapy must be considered, especially if there is marked hypoxia and cyanosis. Although oxygen (100 per cent) has been given for long periods of time, good results are achieved with oxygen in the 70-90 per cent concentration, combined with other gases using a mask for the oxygen alone, or oxygen and air, or oxygen and helium (20 per cent and 80 per cent, respectively), or oxygen (90-95 per cent), with carbon dioxide (5-10 per cent). This latter, however, is contraindicated in patients with pulmonary hemorrhage, severe emphysema, bronchiectasis, acute pleurisy with effusion, and in those with marked pulmonary fibrosis. Mixtures of 75 per cent helium, 20 per cent oxygen, and 5 per cent carbon dioxide have also been used for 5-10 minute intervals with Vaponefrin or Isuprel. I have had no personal experience with the three gases used together.

When the patient's symptoms are more severe, so that he is in status asthmaticus, or in a state of intractable asthma for three or more days, he will almost always require intravenous therapy. From the medical point of view, he now presents a bronchospasm with mucous membrane edema, and inspissated mucus, a cyanosis (mild or severe), and a dehydration with a low blood sugar and low sodium, and occasionally, low potassium values. He may be suffering from an incipient or true right or left-sided heart failure. His asthma is undoubtedly not of the simple type, excepting when due to an overdose of a food or a drug to which he is sensitive, but is more often due to infection, tumor, or degenerative processes, such as a bronchitis deformans.

If the physician attempts to take a history from such a patient, he must remember that the previous drugs may have depressed both the insight and the memory. Such patients not only need 500-2000 ml., or more, of water for heat regulation and kidney function, but general treatment, especially in the middle-aged group in whom other conditions, such as fever, essential hypertension, and occasionally diabetes may be present. In the milder degrees of intractable asthma, when such conditions are absent, water by mouth or isotonic glucose in doses of 1000-3000 ml. within 24 hours may be all that is needed, but if there has been excessive perspiration, nausea, vomiting, or fluid loss due to excessive expectoration, especially if ammonium chloride has been used to excess, or aminophylline or the xanthines have been administered, the dehydration may be associated with low blood sodium levels. If the patient has, in addition, been on a low salt diet for hypertension, he may present the same syndrome which may become more intense if fluids have not been restricted during his present illness. If, as occasionally occurs, there has been a removal of chloride from intestinal secretions as by the Miller-Abbott tube in patients in whom the asthma is a complication of an intestinal condition, such chloride losses must be made up. The usual 0.9 per cent solution of isotonic saline may not be enough, and it may be better to use the 3-4 per cent hypertonic solution in smaller amounts, giving the patient his total normal requirements of three grams daily. If in doubt, Ringer's, Hartmann's, or Fisher's solution should be used.

If the patient has an output of 1500 ml. of urine, with a sodium concentration of 3 gr./L in 24 hours,

he has a normal salt and water balance. For one who requires frequent chloride determinations at home, the Fantus Test is worth knowing. The reagents can be carried in the physician's bag. To 10 drops of urine are added a few drops of potassium chromate. With the same dropper, rinsed, is added silver nitrate in the 2.9 per cent solution, drop by drop until there is a permanent color change from yellow to red-brown. The number of drops required is equivalent to the number of grams of sodium chloride in each liter of urine.

In patients seen at home, or where laboratory studies are not available, the clinical signs of the syndrome are viscid sputum and saliva, diminished integumental turgor, dryness of the skin and mucous membranes, and in more severe cases, a wrinkled cornea and soft, receding eyeballs. If blood counts can be taken, there will be a hemo-concentration and a true secondary polycythemia. The laboratory studies will demonstrate a diminished plasma volume, a high N.P.N., and a high blood nitrogen. In extreme dehydration and hypochloremia, the patient's typical dullness may be masked by sedatives as he progresses through drowsiness to stupor and coma. The classical anorexia and nausea may be masked by the respiratory difficulty, and the muscular cramps and twitching may be lacking in those patients who have received codeine for their cough, or Demerol for their sedation.

Sometimes, in dehydrated patients who have received excessive glucose, more than 12 grams hourly, and in addition, epinephrine by injection, there may be a hypopotassinemia. Taking the normal values as 16-22 mg./L, there are signs of weakness at the 4.0 mg./L level, with respiratory difficulty at the 3.5 mg./L level, and difficulty in swallowing at the 2.5-3.0 mg/.L level. Treatment is by potassium chloride, two grams, or potassium citrate, three grams, every three hours for six doses. If the patient is eating, then foods high in potassium content, such as chicken broth, oatmeal and orange juice should be prescribed. The electrocardiograms, taken in such patients, prove the low potassium levels by the prolonged QT interval; the depressed S-T segment, and the isoelectric, or low, T waves, with occasional prominent U waves.

Similar symptoms occur in hyperkalemia. At the 6.-8.0 mg. Eq/L, the P waves are lowered, the PR interval prolonged; the QRS interval is also widened, with wide, deep S waves; the T waves are tall and wide and may be peaked. In severe conditions, the P waves disappear and the QRS wave is biphasic and bizarre.

Occasional patients present a hyperhydration, which when mild, is characterized by nausea, vomiting, headache, and increased salivation, with weakness progressing to convulsions and coma. Hypertonic saline is necessary, although moderate cases may correct themselves in 6-8 hours without medication.

In patients who have been given morphine, or in whom there may be obstructions of the respiratory passages, or an infection, there may be an acidosis, especially if there has been an insufficient food intake or a great deal of vomiting. The acidosis may also be masked by the severe asthma. The carbon dioxide combining power will be diminished from the normal 55-75 volumes per cent, these normal findings being approximately eight per cent lower in females, and approximately 10 per cent lower in children. If blood chemistry studies are available for sugar and non-protein nitrogen, these may help differentiate such simple acidosis from that of diabetic or renal origin. In these patients, sodium racemic lactate injections, if available, may be life-saving. The dose is 10 ml. of the concentrated solution of sodium-r- lactate/kg body weight. The standard solution is diluted with five times its volume of distilled water before administration. These are the patients, of course, who require oxygen by mask or tent.

In the hyperventilation syndrome of asthmatic patients, rarely, alkalosis may be present. This is also seen in patients given, although rarely, expectorants to excess. Here, the clinical symptoms are headache, restlessness, tremor of the eyelids, and the usual anorexia, nausea and vomiting, with dryness of the mouth, numbness and tingling. The condition progresses, as is well known, to tetany and convulsions. The urine is alkaline and its ammonium chloride content reduced. The blood urea is increased. For these patients, acid sodium phosphate in the one gram dose every three hours, by mouth, is helpful, as is glucose given by mouth and intravenously. In these patients, it is essential that oxygen, if used, be combined with CO_2 up to 10 per cent. If there is respiratory depression and carpopedal spasm, ammonium chloride may be given orally in doses of 10 grams for a total daily intake, or intravenously, in the 0.9 per cent ammonium chloride solution until the symptoms are relieved.

In general practice, the patients seen at home in severe status asthmaticus may, after some days, present a severe hypoproteinemia. The history of having been able to take no food, associated with signs of marked weight loss, dryness of the skin and tongue, and in extreme cases, subcutaneous edema affecting not only the lower extremities and the back, but the face and hands, arouses suspicion of this syndrome. Plasma, which contains not only the electrolytes of sodium and potassium, but sugar, fats, and protein (six per cent) is the immediate intra-

venous solution of choice, followed by food or by injectable amino acids, combined with glucose and plasma for nitrogen balance.

The treatment of any concomitant infection, or other conditions, is a true part of the general treatment of bronchial asthma. Psychotherapy is not suggested in the therapy of any specific attack, although it is usually a part of the general treatment between attacks. During such times when the patient is free of symptoms, general treatment of his asthma can be given due consideration. Studies to elicit the causes and reasons for his wheezing are best done at the office, and during periods of comparative good health. The elimination of causative allergens, the injection of those which cannot be eliminated, medication given for the residual symptoms following elimination and injection therapy, the surgery required to correct abnormalities in the nose and throat, and psychotherapy, all belong at this point in the treatment. All of these are absolutely essential if severe attacks of bronchial asthma are not to recur.

Appendicitis: A Review of 579 Cases*

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Waltman Walters in a discussion of surgical diseases of the abdomen makes the following statement about acute appendicitis: "Acute appendicitis is frequently encountered and all have much experience with it. I shall, therefore, pass it over quickly. The diagnosis is made from the history and physical findings; urinalysis, leucocyte count and a roentgenogram of the thorax are of help in the differential diagnosis. Operation is indicated even when doubt exists."

Such a clear concise statement in this mid-period of the 20th century would seem, actually, to cover all we might need to say concerning the subject of appendicitis. But it was not always thus and it is of considerable interest to think back, briefly, over the beginnings of specific knowledge associated with this most common surgical disease.

Then, we learn of Lawrence Heister who in 1711 described an intraabdominal abscess discovered at autopsy, the abscess apparently having its origin from a perforated appendix. And later, nearly 50 years later in fact, we learn of Mestivier, who demonstrated an abscess originating from a pin in the appendix, the abscess being surgically drained. Throughout the intervening years some 141 cases of disease of the appendix were described in literature until the important paper by Reginald Fitz who described in 1886, 257 cases of perforating ulcer of the appendix, and finally defined the disease. But it fell to the fortunate lot of Thomas J. Morton of Philadelphia in 1887 to deliberately do the first appendectomy. There follow the works of Ochsner, Murphey, McBurney, and Deaver and the improvement in management of things in medicine which have created the excellent surgical care we have today.

It is the purpose of this paper to report a review of 579 cases diagnosed as appendicitis or possible appendicitis and incidental appendices removed at the St. Mary Hospital during the years 1943 to 1948, as tabulated in Chart I.

CHART I

	Diagnoses	*No Report*	*Pathology*	*No Operation Or No Report*
1943				
ACUTE	42	5	34	4
CHRONIC	9	....	18	....
1944				
ACUTE	43	0	32	2
CHRONIC	6	....	15	....
1945				
ACUTE	60	2	51	3
CHRONIC	8	....	18	....
1946				
ACUTE	77	0	51	5
CHRONIC	18	....	39	....
1947				
ACUTE	117	0	70	8
CHRONIC	31	....	70	....
1948				
ACUTE	113	0	79	10
CHRONIC	42	....	66	....
TOTAL				
ACUTE	452	....	317	....
CHRONIC	114	....	226	....

Those of the staff who diagnosed and treated these cases may take all the reflected glory they can absorb—since Murphey, Ochsner, McBurney and Deaver are not with us to challenge. However, we

*Read before the staff of St. Mary's Hospital, Manhattan, Kansas, March, 1951.

must have followed their precepts or those who preceded them for our mortality has been found to be the infinitesimal figure of 0.5 per cent in this series.

And it is interesting further to note that the three cases representing this mortality, which will be discussed in some detail, approach the category of the nearly unpreventable. The mortality, therefore, for the entire series is 0.5 per cent. By years this is tabulated in Chart II.

CHART II

MORTALITY TABLE

Year	*Per Cent*	*Number of Cases*
1943	0	56
1944	0	48
1945	0	72
1946	0	95
1947	0	148
1948	3 cases (1.9%)	159

TOTAL MORTALITY—0.5% FOR SERIES

The first case of mortality associated with disease of the appendix in this series was that of an 89-year-old white woman who had complained of a "sore spot" in the left lower quadrant of the abdomen for three weeks previous to admission. Upon admission she was nauseated and vomited. Physical examination revealed the presence of a diffusely tender abdomen with more tenderness in the right lower quadrant than elsewhere. Rebound tenderness was present. It was decided, because of the advanced condition of the disease, to treat the patient medically and no operation was done. She succumbed and an autopsy revealed the presence of an appendiceal abscess the size of an orange and a perforation at the base of the caecum. There was no general peritonitis.

The second case was that of a 35-year-old white male. He had been under treatment for a peptic ulcer for approximately a year. Thinking his abdominal pain, which he had previous to admission, was due to the ulcer he saw his physician late in the course of the disease of the appendix. His hospital course was stormy from the time of the appendectomy, which was done on admission, through more surgical procedures: a partial bowel resection which included an inflammatory mass and, following an evisceration four days later, a resection of the bowel. The cause of death was reported as "overwhelming toxemia due to hepatorenal syndrome due to inflamed appendix, peritonitis with abscess formation." Total time from admission to death was 12 days.

The third mortality was not directly accountable to the appendix. Complications of polycythemia vera were associated.

The History

Taking as an outline the brief resume by Waltman Walters, the history becomes of prime importance in the diagnosis of appendicitis. Disregarding the possibility of becoming trite in description of a well known disease, the usual listing of onset of appendicitis must be mentioned.

1. *Pain.* Pain is usually the first symptom. It is often sudden and paroxysmal, may occur around the umbilicus or epigastrium and later localize to the right lower quadrant of the abdomen. We remember, however, that there is no such thing as a typical appendiceal pain, as localization of pain will depend on position of the appendix. Unfortunately, in this summary no correlation between onset pain with position of the appendix could be made, for operation reports only infrequently mentioned the exact location of the appendix in relation to the caecum, the peritoneal wall or pelvis. It must be added for completeness, however, that if the appendix lies low in the pelvis or if it is retrocaecal or if it is floating free in the abdominal cavity in respect to the peritoneal wall, the localization of pain and the presence of rigidity are influenced.

All cases in this series diagnosed as acute, numbering 455, had some type of pain a few hours before admission to hospital. Illustrating the fact that the appendiceal history is seldom "typical," 237 cases are reported as having the onset of pain in the right lower quadrant. There were 107 cases of generalized abdominal pain with no localization to the right lower quadrant, 196 cases with general or umbilical-epigastric pain with localization to the right lower quadrant.

The majority of the 124 cases diagnosed as recurrent, interval or chronic presented no uniform pattern as to pain. Many of the patients came in for so-called interval appendectomies, but all recurrent cases presented histories of attacks of pain recurring over a variation of 15 years to the usual history of the previous two years. Soreness in the right lower quadrant and onset of pain in this area seemed more frequent in these groups as well as in the acute cases.

Unusual histories of onset of pain were obtained in the following instances:

a. One patient complained of onset of pain in the right knee which then localized to right lower quadrant of abdomen.

b. Another described a very severe headache followed by onset of pain in abdomen.

c. A sore throat was described as the initial pain. This may be one of the cases of appendicitis of hematogenous cause.

d. One patient reported as having "gas on stomach" and then pain in right lower quadrant.

e. Two patients described a pain in the right hip as initial onset.

f. One patient had no pain but had a weight loss since her baby was delivered 10 months before. Tenderness was prominent over appendix and a pathology report of the appendix was that of an acute diffuse appendicitis.

g. Two patients had pain beginning in the lower left quadrant.

h. One had pain across chest and radiating to abdomen.

i. One had aching under the right costal margin.

2. *Nausea, vomiting and anorexia.* These symptoms are considered as graduations of the same physiological effect of gastrointestinal irritation. Nausea, vomiting, anorexia may be said to be usual but not constant symptoms.

a. 80 cases reported as having definite nausea or nausea and vomiting.

b. 30 cases reported as having anorexia alone.

c. 347 cases: anorexia, nausea and vomiting not reported by the history or the patient did not have these symptoms.

Thus only 20 per cent of entire series presented these symptoms.

3. *Chills or chilly sensations.* Chills or chilly sensations were described by five patients. It is said that chills are more frequent when the appendiceal vessels are initially involved.

4. *Diarrhea.* Diarrhea was present in 14 cases. It is stated in literature that diarrhea in appendicitis is often associated following use of cathartics. No such history was obtained in these 14 cases.

5. *Fever.* Fever was low grade as is expected in early appendicitis. Many had no pyrexia but those who did had fevers ranging from 99° to 103°.

6. *Age Incidence:*

Year	*Number of Cases*
1—10	45
11—20	211
21—30	198
31—40	55
41—50	43
51—60	15
OVER 60	9

It was impossible to find the age of patients in 12 histories. It is obvious that appendicitis is a disease of the young but that no age is really exempt. The youngest case was 11 weeks old. The oldest was 89 years.

7. *Sex Incidence:*

Males	266
Females	307

These figures do not agree with reported experiences that men are slightly more affected by appendicitis than women.

8. *History of previous attacks:* 305 cases reported one or more previous attacks of abdominal pain; 246 reported no previous attacks; 22 histories did not mention whether or not the patient had had previous attacks.

The Physical Examination

1. *The physical findings:*

a. Tenderness in right lower quadrant was reported in over 90 per cent of the cases. Of the remaining 10 per cent the presence or absence of right lower quadrant tenderness was simply not mentioned in the history. Some of these cases had incidental or interval appendectomies.

b. Muscle spasm or rigidity is mentioned in approximately 50 per cent of cases.

c. Rebound tenderness is frequent finding in nearly all of the acute cases.

d. Localized rectal tenderness, although present in most of the acute cases, was infrequently included in the report of the physical examination so no tabulation of this finding can be accurately determined.

2. *The leucocyte count.* The correlation of the white blood count with pathology of blood counts is placed in numerical categories as follows:

	Acute	*Chronic*
5,000 to 10,000	43	95
10,000 to 15,000	74	70
15,000 to 20,000	78	17
OVER 20,000	66	5

Some cliches relative to the white count:

a. A safe point of view is to regard a leucocytosis of 10,000 to 20,000 as confirmatory evidence of appendicitis.

b. Over 20,000 raises the question of perforation.

c. Children's counts tend to be a little higher.

d. A polymorphonuclear leucocytosis may be present with practically a normal total WBC.

Associated Diseases and Conditions

During the patients' stay in the hospital or during the operation many associated conditions were noted:

1. Acute hordeolum—One case

2. Pelvic pathology treated surgically or found during operation in which the appendix was removed. This included such conditions as endometriosis, ovarian cysts, uterus fibromata, uterus suspension operations, salpingitis—20 cases

3. Convalescence from colds and respiratory disease previous to appendectomy—five cases
4. Meckel's diverticula—One case
5. Epigastric hernia—One case
6. Pregnancy at 4½ months, at five months and at eight months
7. Infected diverticulae of colon—One case
8. Rupture Graafian follicle—One case
9. Cholecystectomy—Two cases. This of course represents the gall bladder removed during a deliberately planned appendectomy and not the reverse.
10. Inguinal hernia—Two cases
11. Epilepsy—One case
12. Tonsillectomy—Two cases
13. Vagotomy—One case
14. Pinworms in the appendix—Two cases
15. Terminal ileitis—One case
16. Perforated gastric ulcer—One case
17. Umbilical hernia repair—One case
18. Thyroid adenoma—One case
19. Marginal gastro-enterostomy ulcer—One case

The Operation

In order to determine any change in technic over the five-year period the type of incision and care of the appendiceal stump are tabulated by years. Operating technique in appendicitis is, in general, so well known and so well standardized that very little time is devoted to it. Only an occasional article appears in literature in these years discussing this subject and those discuss unusual or minor changes in operative technique. It is interesting, though, to review our own methods and note the minor changes that gradually take place making the procedure a more successful and safer result.

The only significant change in type of incision used is the increase of the transverse incision after 1944.

There is shown also an increase to 1948 of the use of non-inversion of the appendiceal stump.

Use of drainage material was reported as follows:

In 1943, Penrose drains in three cases.

Between 1943 and 1948, 10 cases.

In 1948, four cases.

Total, 17 cases in which drainage material was used.

The use of antibiotics varied from one to 22 days.

Sulfonamide powder was used in the abdomen during the operation in 227 cases. Penicillin was used once in the abdomen and penicillin and sulfa were used once.

CHART III

POSTOPERATIVE USE OF ANTIBIOTICS

1943	
SULFONAMIDE	9
From one to six days	
1944	
SULFONAMIDE	6
PENICILLIN	1
PENICILLIN AND SULFA	1
1945	
SULFONAMIDE	2
PENICILLIN	6
PENICILLIN AND SULFA	2
1946	
SULFONAMIDE	3
PENICILLIN	14
PENICILLIN AND SULFA	3
1947	
SULFONAMIDE	0
PENICILLIN	25
PENICILLIN AND SULFA	1
1948	
PENICILLIN	32
SULFA	1
STREPTOMYCIN AND PENICILLIN	1

Pathology

The pathologist reported 281 cases as being acute. The diagnosis of acute appendicitis was made 439 times. Therefore, in 208 cases that were diagnosed as acute the pathologist did not concur, but reported some type of chronic condition of the appendix. It is not logical, therefore, to assume that this is gross error in diagnosis. It is, rather, logical to assume that correct diagnoses are made in a much higher percentage of cases than the tabulation of the pathological diagnosis would indicate for the following reasons:

1. The reduction in mortality from appendicitis has in great part occurred because of early treatment and, when ruling out other diseases as best one can, the benefit of the doubt is given in the direction of acute involvement of the appendix and immediate surgical care.

2. Symptoms and signs with reference to the appendix are not pathognomonic, in degree, for an atrophic fibrotic or a lymphoid hyperplastic appendix may have enough acute involvement to produce symptoms identical or nearly identical to its chronic counterpart.

For illustration let us consider in detail the fairly well known pathogenesis of the disease. We may say first that appendicitis may occur in obstruction of the appendiceal lumen, i.e. cicatricial scarring,

foreign body reaction such as fecaliths pinworms, swelling of the mucosa, etc.; second, following respiratory infections; third, hematogenous.

One of the last two conditions in conjunction with the first produces the situation of acute infection and death of tissues in the appendix. Added to this is the fact that the appendiceal artery is a terminal one with no anatomic and swelling pressure if the organ places it in jeopardy by blocking of its blood supply.

Acute Catarrhal Appendicitis

Acute catarrhal appendicitis can almost be spoken of as a "text-book" disease. It is the earliest and mildest form and seldom appears at the specimen table. It must be limited to the following conditions to be so called: mucosa and lumina properia inflamed only. The appendix appears slightly reddened and has a swollen mucosa (49 cases).

Acute Suppurative

This is commonest (210 cases). This occurs as a mild extension from the mild cases just mentioned to a complete disruption of the appendix by gangrenous process with the following characteristics: 1. ulceration; 2. bright cherry red color with ecchymosis through color changes to yellowish, green, dark red to black; 3. swelling; 4. purulent exudates; 5. abscesses; 6. necroses; 7. fibrinous deposition on serosa; 8. perforation.

Another classification of the acute involvement appendix may be made thus: 1. acute diffuse suppurative; 2. acute focal suppurative. Since this series was not reported in this manner it is impossible to classify them.

The subacute appendix begins and continues as a subacute process or recurs from an acute one.

The chronic appendix is described in many ways. The description of course varies as to the pathologic elements present in the appendix.

One interesting phase of pathology of chronic appendix is the lymphoid hyperplasia. Here there is an increase in the lymphoid elements of the organ and it falls into the category of the chronic lymphoid hyperplasia such as seen in tonsils or lymph nodes of the mesentery and it does, in fact, sometimes coexist with lymphoid hyperplasia of mesentery nodes.

The miscellaneous groups are more or less self explanatory.

In consideration finally of the pathology of the appendix and the close integration of the terms used, the process of inflammation varies in degree

SUMMARY OF PATHOLOGICAL DIAGNOSES

ACUTE CATARRHAL		*ACUTE SUPPURATIVE*			
a) MILD ACUTE	(19)	a) ACUTE DIFFUSE ULCERATIVE	(210)	a) GANGRENOUS	(14)
b) ACUTE INTERSTITIAL	(5)			b) GANGRENOUS WITH PERFORATION	(5)
c) CATARRHAL REACTION	(1)				
d) ACUTE HYPERPLASTIC	(1)				
e) RECEDING SUBACUTE INTERSTITIAL	(1)				
f) HEALING	(20)				
g) SUB-ACUTE AND CHRONIC HYPERPLASTIC	(2)				
TOTAL	*(49)*				
SUBSIDE OR OPERATION		SUBSIDE OR OPERATION		PERITONITIS AND OPERATION OR SUBSIDE	

SUBACUTE

CHRONIC (?) OR RECURRENT

CHRONIC FIBROUS		*LYMPHOID HYPERPLASIA*	(18)
a) ATROPHIC FIBROTIC	(7)	a) HYPERPLASIA	(1)
b) SCLEROTIC	(41)	b) HYPERPLASTIC OBLITERATIVE	(1)
c) OBLITERATING	(59)	c) HYPERPLASTIC ACUTE	(1)
d) ATROPHIC OBLITERATIVE	(3)		
e) ATROPHIC	(38)		
f) SCARRED	(28)		
g) FIBROTIC	(1)		

and produces physical findings and symptoms which vary also in degree, but not always in proportion to the acuteness of the process.

For completeness actinomycosis, tuberculosis, and tumors of the appendix should be mentioned. No cases of actinomycosis or tuberculosis were reported. One case of the commonest tumor, the carcinoid tumor, is reported. The carcinoid tumor is almost invariably non-cancerous.

Chronic Appendicitis

The clinical interpretation of chronic appendicitis is a much greater problem than that of acute appendicitis. The diagnosis seems to be made frequently and, when made, is difficult to prove even when the appendix is studied by the pathologist.

A summary of pathological diagnoses is based on a supposition that the various forms of chronically involved appendices develop their conditions as a result of previously occurring acute involvement either as repeated attacks of acute inflammations or a gradual process of inflammatory changes progressing to the final chronic state. Many have questioned this pathogenesis. Especially has it been questioned in regard to the fact that fibrosis, being an end result of inflammation, is not an indication that the inflammatory reaction exists at the time the surgeon removes the appendix. In other words, it is difficult to correlate the patient's symptoms of dyspepsia, nervousness, poor appetite, etc., as being due to a chronically fibrosed or obliterated appendix in which the signs of inflammation have subsided.

CHART IV

APPENDICITIS

Age	*Keith*	*This Series (Chronic Obliterative)*
10 YEARS	4% (2)	3
20 YEARS	7% (15)	27
30 YEARS	17% (33)	18
40 YEARS	25% (14)	9
60 YEARS	36% (5)	2
70 YEARS	50% (4)	2

Some theorists such as Sir Arthur Kieth, Ribbert, Kelly at Johns Hopkins and Boyd in the Winnipeg Hospital believe that these atrophic changes are a result of more or less natural retrogressive processes associated with the advance in years.

One very interesting feature of the development of the chronically involved appendix is the concept of Masson regarding the hyperplasia of non-medullated nerve fibers found in the submucosa of these appendices which may also contain many small circumscribed neuromas. The muscularis coat may also contain a marked hyperplasia of Auerbachs plexus with numerous ganglions with cells several times larger than normal. Further study may reveal in the future that nerve tissue hyperplasia accounts for symptoms associated with these chronic changes seen in the appendix.

CHART V

COMPLICATIONS

WOUND DRAINAGE AND WOUND INFECTIONS	25
HYPERPYREXIA TEMPERATURE OVER 103° p.o.)	8
HEMATOMA OF WOUND	2
HORDEOLUM	1
SEVERE SULFONAMIDE REACTION	1
PHARYNGITIS	1
CHILLS (p.o.)	1
MUMPS	1
MENTAL CONFUSION (p.o.)	1
SEVERE VOMITING AND ABDOMINAL PAIN (c.o.)	1
DIARRHEA	1
PNEUMONIA	2
DIABETES	1
HEART DISEASE	2
MARGINAL ULCER GASTRO-ENTEROSTOMY	1
BOWEL RESECTION, EVISCERATION	1
TOTAL COMPLICATIONS	50
PERCENTAGE	9.6%

Summary

Appendicitis occurring in 579 persons between the years 1943 and 1948 inclusive has been studied. They include a mortality of 1.9 per cent for the year 1948 and a mortality of 0.5 per cent for the series.

Fourth Annual

MID-WEST CANCER CONFERENCE

April 3 and 4, 1952 Broadview Hotel, Wichita

BLUE SHIELD

The Quiet Approach

The gist of a recent editorial in the *Lawrence Daily Journal-World* was that doctors should not relax their efforts to inform the American people on the advantages of private medical practice over a compulsory governmental program. Commenting on a recent announcement that the American Medical Association was relaxing its campaign against socialized medicine because the battle has been won, the *Journal-World* said in part:

"It behooves the doctors to quietly continue to sell the public on the advantages of private medicine over government operation of all offices and hospitals. It is up to the doctors to make the present plan work efficiently and satisfactorily for the public good."

The golden opportunity for the ultimate victory of free medicine lies in the years just ahead. The job of "quietly selling" the public must be done through deeds as well as words. As delightful as it might be for doctors to forget the economic problems of good medical care so that all of their energies might be devoted to its practice, the time is still not right for such a luxury. There is still the need for a positive selling job even though some of the hue and cry and feverish excitement have died down.

The economic problem is still present and could easily become much more acute as inflation continues. In Kansas, for example, less than half the population is covered by some form of hospital protection. An even smaller percentage has protection against the cost of medical care. At least 85 per cent of our people need this kind of protection, but many still do not understand the problem.

A positive program on the economic side would be a greater promotion of Blue Cross and Blue Shield. We like the phrase "quietly selling." It fits in with the approach Blue Cross and Blue Shield have tried to follow. This year more than ever we are asking the doctors and the hospitals to help in bringing about a greater public understanding of the problem and of the positive way it can be met. This will not require flamboyant claims, overstatements and scare headlines. It can best be done through complete honesty with the public, through contracts that deliver what they say they will deliver, and through sincere identification of the doctors and hospitals as the originators and backers of the Blue Shield and Blue Cross program. It can be done by talking with people around the table, by meeting with them in community groups.

The years ahead will give us time for the quiet approach. Through the very plans which the doctors and hospitals have created, we can find most of the answers to the economic problems of good health care. This can be done with one main proviso: If the medical profession will continue its interest in the problem and will actively participate in the solution.

SOCIALIZED MEDICINE

This completes a series on the subject of socialized medicine. Twenty-three separate items have been published on this subject presenting factual information both from the proponents and opponents of socialized medicine. It is hoped that compilation of this material will provide physicians who are requested to write or speak on this subject with illustrative data.

At the moment advocates of socialized medicine are still active. The recently adjourned Congress saw numerous bills designed to further one or another phase of this program. There is no lull in the pressure that is being brought to bear. However, in the interim between sessions of Congress, there will be no new activity in this field and for that reason this series will be discontinued until further legislative attempts arise.

Laboratory Program Announced

The College of American Pathologists has announced a program to improve the efficiency and to increase the uniformity of medical laboratory analysis. The initial step will be the supplying of highly standardized and very accurate solutions to large and small pathology laboratories to develop and maintain uniformly reliable test solutions in the field of chemical determinations.

College members, who represent about 80 per cent of all certified pathologists, now have a uniform source of standardized, accurate chemical solutions prepared in such a manner that deterioration is unlikely. These solutions can be used to verify the correctness of the individual laboratory stock solution, to check the accuracy of personnel in performing a test, or to serve as a daily reference standard when determinations are made.

Pathologists have taken the lead in an attempt to improve the accuracy of laboratory tests. The first step, dealing with the standardization of the Wassermann test, was taken by the American Society of Clinical Pathologists before the College of American Pathologists was organized. Before that time, samples of the same blood tested in different laboratories sometimes produced contradictory results.

PRESIDENT'S PAGE

Dear Doctor:

At the present time we find the complexities of living increasing by the day. Tension has so steadily been mounting that many of us have felt the breaking point to be just around the corner.

The complexities, tension, taxes, et cetera seem to have clouded our vision and thinking to the point that recognition of friendly assets and the maintenance of balance in reason have at time lacked appreciation. It is gratifying, however, to find that many still possess the balance, the reason, the clarity in thinking and continue to recognize normal allied relations!

I am glad to report to each of you I have been privileged to talk with officials of the professional societies of the doctors of dentistry, of hospital administrators, nurses, pharmacists, and of veterinary physicians and surgeons. I find we are in essence, of singular thought and opinion!

That opinion is that we are all interested in the health and protection of Kansas citizens and we are all closely inter-related, yet in confusion of purpose and lack of organizational understanding each of us has experienced some myopia and has overlooked the forest on account of the trees. Recognition of errors with proper adjustments is truly evidence of substantial progress!

I am proud to report to you that in my humble opinion each of our normal allied professional groups displayed equity, courage, and balance and gave evidence that was positive in character that they were acutely aware of their individual responsibilities as well as our joint obligations. To me the unanimity was most wholesome and stimulating.

I proposed a committee without official capacity (wholly advisory) composed of the allies with equal representation for the purposes of unity in striving for better government, unity in improving the public's health, unity in seeking better economics, better understanding of our social problems and manifestations of political strength in seeking to maintain the principles of true Americanism, one of which is private enterprise!

It is with pride I report to you that your Council approved the proposal on November 4, and may I suggest you seek the details from your Councilor and furthermore take steps to institute the machinery on local, county and community levels. Proof of the pudding is in its tasting!

In conclusion, I should like to emphasize that your support and participation in your society will render you excellent dividends today and posterity will record you as a worthy member of the greatest of all professions.

Very truly yours,

O. H. Benage

EDITORIAL COMMENT

A Fable

A strong young man came into the wilderness, broke it open and made a farm. Soon others around him began to till their land until he was the center of a growing community of pioneers. They all prospered until barbarians began to raid and plunder the possessions of those on the periphery.

In a short while the outlying farmers became hungry because their crops were continually destroyed, so the strong young man whose land had not been molested shared his abundance with those who were less fortunate. For a time they all survived in this way. The farmer in the center was not altogether altruistic. He recognized this as insurance against the destruction of his property so he was satisfied.

Before long the farmers at the wilderness fringe recognized this also. They saw here an opportunity for capitalizing on their misfortune so bargained for more of his goods. Becoming alert to this possibility they organized against him. Their motive was not altogether destructive for they united in their common welfare. Therefore as a unit they presented a logical plan to the strong young man in the center who accepted it and paid higher rates for his protective insurance.

His one farm now had to provide food for those living on many farms. He gradually discovered that they no longer planted seed on their land but were content to exist on what he gave them. Nor did they expect to pay him because they reasoned that the protection he had from them was payment enough. It was not that his neighbors were essentially lazy. It was only that experience taught them the futility of effort and the contract with this young man made effort unnecessary. So things rocked along for a while without any great crisis, but not forever.

Something else began to happen. No one can tell exactly how this all came about or what was first because history crowded the stage. Suddenly everyone began to feel differently and all previous contracts came apart like this:

The outlying farmers, no longer in fear of losing their crops, now had only to protect their lives. This, being a serious business, soon reacted to where they and their savage attackers became acquainted. As they saw more of each other an understanding grew up and finally these farmers and their enemy made their own agreement. In return for certain considerations there would be no further attacks. It was that easy.

Of course the considerations involved the strong young man in the center who was then in the dilemma of finding those he had benefitted were now united with the enemy and in this bizarre situation he would pay both. To do so was bankruptcy, to refuse was suicide, so he did the only thing he could do. He also abrogated his former agreement and secretly made a deal directly with his principal enemy, the savages. By ignoring his former friends he had only to satisfy the attackers, which he did.

And that set the stage for the end of the fable. The outlying farmers were now without food for themselves, nor could they provide the considerations required by the enemy, so they were destroyed. Then the strong young man found himself without allies. The attackers made more and heavier demands. When it became altogether impossible to comply, the enemy moved in and killed him also. In a very short while the savages with no one else to plunder began fighting among themselves and the farm was soon a wilderness again.

Eye Testing for School Children

Visual defects in school children, long recognized as factors contributing to emotional, social and educational maladjustments, have been cared for or not according to the attitude of parents. A plan for the systematic search for children with eye defects and the correction of these conditions has recently been developed by the Committee on Conservation of Eyesight.

The value of such a service is immediately apparent and has been, of course, for a long while, the only deterrent being the lack of a practical method of accomplishing the desired result. This, it is believed, has been found by the committee and is now recommended to the profession.

Visual defects in children can easily be discovered by any intelligent adult who has considerable contact with children. By way of example, the school teacher will learn within the first month which of her children are unable to read what is written on the blackboard. Therefore, the recommended program is not only simple but is also convenient and inexpensive.

Sight defects can readily be determined on a mass testing basis through the use of the ordinary Snellen chart. After a minimum of instruction which is available through the medical social worker of the Services for the Blind Division of the State Board of Social Welfare, the average teacher can give the Snellen test to her pupils. This can be given during school periods with a minimum loss of classroom

time, and is efficient enough to indicate all children with pronounced disorders in vision. Once this knowledge is obtained, a letter may be sent the parents advising a more complete examination and correction wherever that is deemed advisable. They need only to be told that a visit to their family physician is recommended.

The above plan is presented as a potential public service project for county medical societies. If the county society will make the necessary arrangements with school authorities, including acceptance of the program and the permission to utilize the services of a teacher or school nurse for giving the tests, the Kansas Medical Society will then make available at no cost an instructor who will advise the teacher on recommended procedures. That is all there is to the program except for follow-up work. This, quite naturally, is dependent upon the enthusiasm with which the project is undertaken and also will control its success or failure.

One other possible approach has been splendidly demonstrated through the efforts of W. H. Walker, M.D., of Eskridge, Kansas. He completed this program for all schools in Wabaunsee County, undertaking it as a project of the Eskridge Kiwanis Club. It could be duplicated by any service club in other cities. The project requires no expenditure of funds, is easily administered, and carries with it not only a public relations value to the sponsoring agency but great community and individual service.

Distribution of Physicians

The so-called Kansas Plan for providing rural areas with physicians has been in operation for several years. During this period, some definite results have been noted. In the years 1900 to 1942, the latter being selected as the last year prior to the war, small towns were consistently losing doctors. There are instances of communities supporting five in 1900 that had none in 1942. During the period of 1948 to 1950, 69 physicians located in Kansas communities of 1,500 population or less. This represents the first reversal of a trend that had been in progress for 50 years.

The Kansas Plan of providing rural areas with medical care is far from completed, but considerable progress has been made. Many smaller communities have provided hospital facilities and office space for physicians, more often than not without the use of federal funds, and shortly thereafter found a doctor. It is significant, if not surprising, that before long the physician felt the necessity to have a second doctor to help him. There are already instances of a third coming to a community which a few years ago had no resident medical care. Therefore, even though there are still many areas that can well support a physician, and even though many more physicians will be welcomed by the medical profession and by the communities involved, the principal factors of the first phase of the problem have been solved.

Repeating again for the purpose of emphasis, many more doctors are needed, but Phase II is already becoming apparent and some concerted action will be needed to cover this portion of the problem. Many communities in this state are asking for medical care which, for one or more reasons, could have better medical attention through the use of a doctor in a neighboring town. A blunt if not altogether accurate condensation of this problem might be made as follows: It is only logical that any scarce commodity or service should be utilized with maximum efficiency. A doctor's maximum efficiency is reached in the hospital. Therefore, his services can best be utilized if patients come to him. The smallest towns require that he spend much of his time in a car visiting farm homes in the surrounding area, therefore will use a doctor at less than the best service of which he is capable. It follows then that there are areas in which two additional doctors in a larger town with a well equipped hospital can serve the two outlying communities more efficiently than if they located in these smaller places.

To prevent the possibility of misunderstanding on this point, this plan might be repeated in other words. Under a free enterprise system the doctor, like the lawyer or hardware merchant, will locate where he wishes to go. The Kansas Medical Society will certainly never attempt to prevent a doctor from locating in any small community. It is believed, on the other hand, that if some of the smallest of these towns that are located near larger cities would cooperate as districts in obtaining medical care, they can find a more efficient and a more economical solution than to build a competing hospital and attempt to support a resident doctor.

In the interest of providing medical care of high quality within easy access to all persons of the state, the above can represent a solution which is both practical and possible of attainment. Modern transportation and paved roads bring the doctor, 30 miles from the rural resident, as close as the average doctor is to the family living at the edge of our larger cities. This is stated crudely and only in part and represents an outline of what should be Phase II of the Kansas Plan. Ideally it consists of having all people in the state understand how the services of a physician may best be utilized. In that way they will obtain the most service at least expense for themselves, and the physician will be rendering the greatest benefit with the least loss of time.

Research at K. U. Medical Center

Within a few weeks a most attractively prepared booklet describing the research projects at the University of Kansas School of Medicine will be available for distribution. This book is beautifully illustrated and well written to give a brief but concise description of what is being done in research at this school.

The book is prepared for public consumption. It is in no way scientific or is it intended to be. By way of example, it lists cancer as one of the areas in which research is being conducted and states in one short paragraph that information is needed in the fields of early diagnosis and new methods of treatment. The pamphlet discusses the high cost of research and makes a dignified and conservative suggestion that the public is permitted to assist by donating money to these departments.

Upon completion a copy of this book will be mailed to each physician in Kansas with the recommendation that it be placed in his waiting room. It is hoped that each doctor will give some care to the book when it arrives so that it may continue to be read by patients in the waiting room.

Vocational Rehabilitation

A committee to assist in vocational rehabilitation in the state was recently appointed by Dr. C. H. Benage, president of the Kansas Medical Society. The following were named as members: Dr. O. W. Davidson, Kansas City; Dr. Dwight Lawson, Dr. Ralph R. Preston, Dr. John L. Lattimore and Dr. W. L. Beller, Topeka; Dr. John A. Grove, Newton; Dr. John M. Porter, Concordia; Dr. Murray C. Eddy, Hays; Dr. G. R. Hastings, Garden City; Dr. John B. Jarrott, Hutchinson; Dr. Conrad M. Barnes, Seneca; Dr. H. H. Jones, Winfield; Dr. E. A. Smiley, Junction City; Dr. Haddon Peck, St. Francis; Dr. N. E. Melencamp, Dodge City; Dr. A. L. Ashmore, Wichita.

The state rehabilitation service assists the disabled of Kansas to become employable by offering vocational counsel and guidance, physical restoration, training, tools and supplies, placement, and follow-up. The duties of the Professional Advisory Committee will be to review the physical restoration work, and to establish fee schedules for medical and surgical treatment, hospitalization, and artificial limbs. The committee will also recommend to the rehabilitation service policies concerning physical restoration and will act as liaison between the medical profession and other groups interested in rehabilitation services.

The rehabilitation of the handicapped is a subject of great interest to the medical profession, and many physicians have cases to be referred to the rehabilitation service. Arrangements for service and requests for speakers for county medical society meetings should be addressed to Rehabilitation Service, 908 Topeka Boulevard, Topeka, Kansas.

Student Journal to be Published

The first issue of the *Journal of the Student Amercan Medical Association*, a 72-page publication, will be published in January as the official organ of the Student American Medical Association organized a year ago.

The journal will be issued nine months of the year, omitting July, August, and September. It will be sent to 26,191 medical students and approximately 7,000 interns.

The space will be about equally divided between editorial and advertising copy, with editorial material including scientific and socio-economic articles, a news letter, information on new pharmaceuticals, equipment, and instruments, book reviews, a question and answer section, editorials, and feature stories.

Club of "Guinea Pigs" Formed

Using the tentative name of the "Guinea Pig Club," a group of scientists met recently in Chicago to formulate an organization which will be comprised of human "guinea pigs." Membership will be limited to medical scientists, students, technicians, and volunteers who have ever served in any way as human test material.

The plans are to formally organize chapters in each medical center throughout the country. The purpose of the organization is for education, and at each meeting papers will be presented describing experiences and results of test service. Citations or some form of honorary recognition for outstanding volunteer service will be presented. Another activity of the group will be to publicize the codified rules now in effect for the conduct of experimental procedures on human beings.

Suggestions on politics, program, and organization details will be welcomed. Communications may be sent to the National Society for Medical Research, 185 North Wabash Avenue, Chicago 1, Illinois.

Cancer Grant to K. U.

The University of Kansas School of Medicine last month was given a grant of $27,635 for the evaluation and development of serodiagnostic tests for cancer from Public Health Service funds allocated by the National Cancer Institute. The work will be conducted under the direction of Dr. R. E. Stowell and Dr. J. H. Hill.

Case Reports from the University of Kansas Medical Center Tumor Conference*

Exfoliative Cytology in the Diagnosis of Cancer

Edited by R. E. Stowell, M.D., and D. M. Gibson, M.D.**

Most authorities agree that the diagnosis of malignancy by exfoliative cytology is a valuable adjunct when used in connection with other diagnostic methods. The following three cases will show some of the benefits to be derived from, and the pitfalls to be avoided in, the cytologic diagnosis of cancer.

Case No. 51-95

J. W., a 66-year-old man, was admitted to the University of Kansas Medical Center on August 18, 1951, with a history of anorexia, weight loss of 25 pounds and a feeling of fullness in the upper abdomen and chest for two months. He had noticed some associated dyspnea and orthopnea. The patient's left chest had been tapped on two occasions 6 and 10 weeks previously with removal of much bloody fluid, said to contain malignant cells. He has been constipated for one to two years, but has had no other gastrointestinal symptoms. He has developed a slight cough during the last two months but has had no hemoptysis.

The patient was not in acute distress. There was dullness to percussion in the entire left chest; the right chest was essentially negative. All cardiac dullness was heard behind the sternum and in the right chest.

Urinalysis showed a slight trace of albumin and a few hyaline casts with 0-2 red blood cells and 0-4 white blood cells per high power field. Complete blood count, blood chemistry, total and fractional proteins and gastric analysis were within normal limits. The electrocardiogram showed left ventricular strain.

A bronchoscopy done on August 20, 1951, showed no abnormalities. Four successive thoracenteses were performed, with the removal of 5,400 cc. of bloody fluid. The patient was discharged on August 24, 1951.

Dr. Dunham: Roentgenograms of the chest taken on admission reveal a massive left pleural effusion. Following thoracentesis, in addition to a hydropneumothorax, a vague pulmonary mass is seen anteriorly. Intravenous pyelograms show definite distortion of the calyces of the left kidney, suggesting a renal tumor. The gastrointestinal series was negative except for sigmoidal diverticuli.

Dr. Pollak: Smears of the pleural fluid show large numbers of bizarre cells, containing large hyperchromatic nuclei and prominent cytoplasmic vacuoles. These cells (Figure 1) were regarded as being malignant cells, probably from a primary carcinoma in the gastrointestinal tract.

Dr. Firminger: Special stains, such as the periodic acid-Schiff and mucicarmine stain, would confirm the presence of mucin in these cells.

Dr. Helwig: Do you think these could be metastatic cells from a renal cell carcinoma?

Dr. Pollak: That possibility certainly can't be eliminated, but the general impression was that the cells were probably metastases from an adenocarcinoma of the gastrointestinal tract.

Dr. Helwig: This man had convulsions that antedated the chest symptoms. Renal cell carcinoma frequently metastasizes to the lung and the brain. Dr. Williamson, do you think this patient has metastases to the brain?

Dr. Williamson: This man had typical epileptic seizures for seven years, which had not changed since the onset of his present illness. The neurological findings were completely negative. The con-

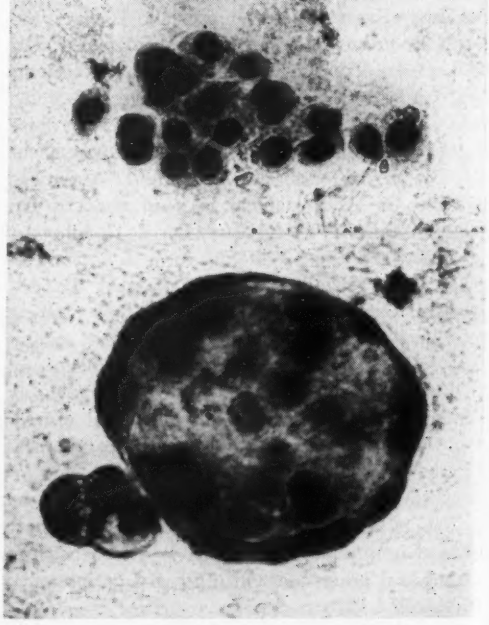

Figure 1. Groups of malignant cells in sputum from Case 51-95. The larger group represents an acinus of cells.

*Cancer teaching activities aided by a grant from the National Cancer Institute, U. S. Public Health Service.

**Damon Runyon Clinical Cancer Research Fellow.

vulsions were always nocturnal and subsided with medication. In addition, one brother and one nephew have epilepsy. For these reasons, we considered the convulsions to be completely unrelated to malignancy, either primary or secondary.

Dr. Kittle: I recall a case similar to this in which malignant mucoid containing cells in the pleural fluid were subsequently shown to be due to a primary carcinoma of the lung, which could not be demonstrated by bronchoscopy. I think a primary carcinoma of the lung should certainly be considered in this case.

Dr. Johnson: A series of examinations of stool specimens for occult blood is a useful method of screening for gastrointestinal cancer.

Dr. Helwig: Some success has also been obtained from surface smears of the colon following a cleansing enema.

There have been several series of cases, including that reported by Alexander,[1] of patients with renal carcinoma and other malignancies with solitary lung metastasis, who have had a long arrest of the disease following removal of the primary tumor and resection of the solitary metastasis.

There have also been quite a number of cases of renal cell carcinoma in which solitary bone lesions have been removed and patients have done well for some time. The peculiar tendency of hypernephromas to yield a solitary metastasis is well known. A hypernephroma is the first thing to consider with a solitary metastasis in the lung. It is important to remember that a secondary pulmonary lesion may also invade a bronchus. I have seen three such cases in recent years. So the fact that a main bronchus is invaded does not mean that the patient must have a primary bronchial lesion.

In summary, this is the case of a man with malignant cells in the pleural fluid. The diagnostic question is, where is the primary tumor. The final diagnosis is not yet available, but one must strongly consider a primary focus in the kidney, lung or gastrointestinal tract. In this regard, an excellent clinical impression to remember is that bloody pleural fluid in an afebrile patient in the cancer age is due to cancer until proven otherwise.

Case No. 31-96

P. I., a 35-year-old man, was admitted to the University of Kansas Medical Center on June 17, 1951, with the chief complaint of fullness in the stomach and moderate dyspnea which began one month before while swimming. This gradually became worse and two weeks ago he consulted a local doctor. He was told that he had fluid around his heart and hospitalization was recommended. A pericardial tap was done and 50 cc. of sterile fluid was removed. He immediately felt better, but the fluid returned and a second tap was done in two weeks with little relief. The day before admission the patient had a temperature of 101.6°. Review of symptoms reveals a sensitivity to novocain and sulfa drugs.

Physical examination of the chest revealed equal expansion bilaterally and percussion dullness posteriorly from the fourth rib downward. Vocal fremitus and breath sounds were decreased over the left posterior chest. The area of cardiac dullness extended into the axilla.

Urinalysis revealed a trace of albumin with many mucus threads. The red blood count was 5.9 million, the white blood count 8,000 and hemoglobin 15.6 grams. The differential count was normal. The blood non-protein nitrogen was 42.5 mg. per cent, creatinine 1.4 mg. per cent, sugar 83 mg. per cent, sodium 142 milliequivalents, potassium 4.7 milliequivalents, chloride 104 milliequivalents. Histoplasmin skin test was positive. Agglutination test for B. tularensis was positive, 1:20. Three successive blood cultures grew B. subtilis.

The patient was placed on dihydrostreptomycin because of the positive blood cultures. Pericardial tap on June 12, 1951, revealed numerous clumps of poorly outlined cells with large vesicular nuclei that showed considerable variation. These were initially regarded as being metastatic adenocarcinoma cells.

On July 9, 1951, an exploratory thoracotomy was performed and the pericardial sac was explored. Approximately 600 cc. of bloody fluid was aspirated from the pericardial sac. Along the course of the anterior descending branch of the left coronary artery there were four or five small nodules varying from one-half to one cm. in size. Because of the relation to the coronary artery, these nodules were not biopsied.

Because of the possibility that the patient had periarteritis nodosa, he was given ACTH, and showed marked improvement. The left hydrothorax and the hydropericardium improved on ACTH and antibiotic therapy and the patient was discharged improved on August 9, 1951.

Dr. Dunham: Roentgenograms taken on admission show a hypertrophied heart. There is also an opacity in the left base that may be interpreted as fluid and pulmonary edema. No cardiac pulsation was noted, which is consistent with pericardial effusion. Subsequent films showed decrease in heart size and feeble pulsations, as well as moderate fluid in the left pleural cavity.

Dr. Pollak: The pericardial fluid showed numerous large clumps of cells, many of which show moderate variation in the size and shape of the nuclei. There is also some variation in the intensity

of staining, and a few large cytoplasmic vacuoles are present. This was first considered as metastatic adenocarcinoma to the pericardium. Dr. Kerr reviewed the smears and felt that these were reactive mesothelial cells, and not malignant cells.

A biopsy of pericardial tissue removed at operation shows groups of cells similar to those seen in the smear. They are iron-containing mesothelial cells grouped in sheets, nests and anastomosing cords. Some of these cells show bizarre size, shape and staining.

Dr. Allen: This case was quite a diagnostic problem from a clinical standpoint. The most important feature was a persistent pericardial effusion in a young, husky, afebrile individual. The positive blood cultures were only obtained in the broth and never in the plates, so this was discounted.

We were faced with the problem of doing exploratory surgery on the patient or giving him x-ray therapy on the assumption that he had a pericardial malignancy. When we learned that the suspected malignant cells in the pericardial fluid were actually non-malignant mesothelial cells, we decided the patient should be explored.

At operation, the nodules that were seen surrounding the coronary arteries were identical to those present in a patient who died at this hospital last year with widespread lesions of periarteritis nodosa. We had included periarteritis nodosa in the differential diagnosis of a patient with bloody pericardial fluid but had never given it serious consideration.

An interesting feature is that this patient raised turkeys. Exploring the possibility of an occupational disease, we learned that a disease of turkeys sometimes produces nodular lesions on the heart, but there is no record of such a condition having occurred in humans.

With the presumptive diagnosis of periarteritis nodosa, the patient was treated with ACTH. He responded very well and is now well, working every day and still raising turkeys.

Case No. 51-97

J. F., a 64-year-old white man, was admitted to this hospital on August 8, 1951, and expired on August 18, 1951. He had been perfectly well until four months prior to his admission, at which time he first complained of weakness and fatigue. Two months before his admission he developed a cough, which was productive of a large amount of whitish sputum, and some pain in the lower portion of the right chest. He developed shortness of breath about one month prior to admission, and noted reddish sputum for the first time.

Physical examination revealed inspiratory and expiratory moist rales and rhonchi over the right lower lobe of the lung and an area of dullness in the posterior right chest. The remainder of the physical examination was essentially negative. The blood pressure was 130/80 and his pulse rate on admission was 80.

Urinalysis showed three to four red blood cells and eight to nine white blood cells per high power field. Blood counts and chemistry were within normal limits. The electrocardiogram shows partial right bundle block and a rate of 140. P waves were large and resembled those found in right or in auricular hypertrophy or dilatation.

The patient became much more dyspneic on the fifth hospital day and there was a marked tachycardia with a rate of 140. He was digitalized the following day but failed to respond. The patient was treated symptomatically. The chest became completely filled with rales, the volume of bloody sputum which he produced became much more marked, and he went into shock and died.

Dr. Dunham: Chest roentgenograms reveal multiple, confluent, poorly defined masses scattered diffusely throughout both lung fields. Pulmonary adenomatosis, although a rare lesion, should be considered in the differential diagnosis, as should lymphogenous spread of a metastatic carcinoma, and an unusual mycotic infection. Gastrointestinal series and pyelograms were negative.

Dr. Boley: Examination of the sputum specimens reveals many groups of cells showing variation in nuclear size and shape and irregular clumps of chromatin. There is some cytoplasmic vacuolization. These are definitely malignant cells.

Dr. Helwig: Dr. Delp, will you discuss this patient from a clinical standpoint?

Dr. Delp: This patient presented signs and symptoms of marked pulmonary insufficiency, probably primary, and cardiovascular disease, probably secondary. The history made it obvious that this was not an acute cor pulmonale (pulmonary disease secondary to embolization), nor did the condition resemble a chronic cor pulmonale, for ventilatory and pulmonary symptoms were absent prior to the onset of the acute illness.

Chronologically, at least, the patient would fall into the category of sub-acute cor pulmonale. This is a term intended to encompass those patients who have pulmonary disease secondary to metastatic lesions in the lung. With diffuse lymphogenous spread of the malignancy, the pulmonary vessels and bronchi are encroached upon, with resultant decrease in ventilation and increase in pulmonary pressure, resulting in cor pulmonale.

Coccidiomycosis and histoplasmosis should be

considered in the differential diagnosis, as should pulmonary adenomatosis, but I thought metastatic carcinoma the more likely.

Our efforts were directed toward trying to discover the location of the primary lesion in a patient who was in critical condition throughout his hospital stay.

Dr. Helwig: As this patient was followed to necropsy, may we have the autopsy findings?

Dr. Boley: At autopsy this patient had a primary malignancy in the lung, apparently of multicentric origin. The cells of the tumor nodules line the alveolar walls. These cells are tall columnar in type and show considerable variation in size. The lesion is present in the hilar lymph nodes. No primary focus has been discovered in the bronchi or in any other part of the body, so we are evidently dealing with an alveolar cell tumor of the lung.

Dr. Helwig: Dr. Kerr, will you discuss the diagnosis of malignancy by cytologic technics?

Dr. Kerr: Exfoliative cytology is a valuable adjunct to tumor diagnosis. Exfoliated cells may be examined from chest fluid, abdominal fluid, vaginal secretions, bronchial secretions, collections of urine, or material from ureteral catheterization. This procedure is not new, but has been in use for many years. Fluid may be centrifuged, and the sediment may be made into paraffin blocks, or the fluid may be examined by smearing it on a slide. Another technic is the sponge biopsy introduced by Gladstone,[2, 3] in which a gelfoam sponge is placed over a lesion, as used to obtain bronchial or gastric secretions. The smear or sectioned sediment may be stained with a variety of technics, depending on individual preference. I prefer the Papanicolaou stain because it gives better nuclear detail.

One important thing to remember, illustrated by the second case, is that any inflammatory reaction involving a serous surface will produce reactive changes in the lining mesothelial cells, so that they may appear bizarre and atypical. Mesothelial cells may be seen in glandular arrangement occasionally, secondary to inflammation. It is amazing that these atypical mesothelial cells are not regarded as being malignant cells more often. Saphir[4] has described the marked atypia that may occur in non-malignant cells in various body cavities.

The cells seen in the pleural fluid in the first case are definitely malignant cells. I think it would be impossible to eliminate a primary carcinoma of the lung in this case, because mucoid carcinoma is not too uncommon in the lung. The tumor may be primary in any structure originating from the primitive gut tube, because these cells are all potentially capable of producing mucin.

In examining various body fluids, I think it is advantageous to study both smears and embedded sediment from each case. Although both are similar in a fair percentage of cases, occasionally one may be considered positive and the other negative. We should use every method at our disposal in attempting to arrive at a definite diagnosis.

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ACTIVITIES OF MEMBERS

Dr. Karl Menninger and Dr. R. C. Anderson, Topeka, were invited by Governor Stevenson of Illinois to attend a meeting in Springfield last month to plan improvements for the Illinois state hospital system.

* * *

Dr. J. P. Haigler, Hays, was principal speaker at a meeting of the Western District of the Kansas Society of Medical Technologists held in Hays last month. His subject was "The Leukemias."

* * *

Dr. Howard Snyder, Winfield, spoke at a convention of the Kansas Federation of Licensed Practical Nurses, October 18 and 19.

* * *

Dr. Ernst T. Morch, assistant professor of surgery at the University of Kansas Medical Center, presented a paper on "Geriatric Anesthesia" at the International Gerontological Congress in St. Louis recently.

* * *

Dr. Mary T. D. Glassen, Phillipsburg, was one of the speakers at a conference held in Wellington last month by the Kansas Congress of Parents and Teachers.

* * *

Dr. Francis X. Lenski, Jr., Iola, is now attending postgraduate courses at the Cook County Graduate School of Medicine, Chicago.

* * *

Dr. D. V. Conwell and Dr. C. J. Kurth, Wichita, attended a meeting of the Central Neuropsychiatric Association in Minneapolis, Minnesota, October 19 and 20.

* * *

Dr. Philip W. Morgan, Emporia, was elected president of the Kansas Heart Association at a meeting held in Emporia last month. Dr. Lee H. Leger, Kansas City, was named president-elect.

Dr. T. H. White, who has been associated with the Student Health Department at Kansas State College since 1946, has entered private practice in Manhattan in association with Dr. Darrel L. Evans.

* * *

Dr. J. J. Hovorka, Emporia, was guest speaker at a Plymouth community meeting last month, giving a talk on cancer.

* * *

Dr. Paul H. Lorhan, of the University of Kansas Medical Center, was one of the instructors at a refresher course given by the American Society of Anesthesiologists, Inc., in Washington, D. C., November 5-7. His subject was "Geriatric Anesthesia."

* * *

Dr. Ray T. Parmley, Wichita, was named an associate examiner of the American Board of Anesthesiology recently. The board met in Memphis, October 14-17.

* * *

Dr. Forrest L. Loveland, Topeka, has been named a consultant for the Southwestern Bell Telephone Company's employees' benefit committee.

* * *

Dr. Franklin D. Murphy, chancellor of the University of Kansas, was speaker at a meeting held in Overbrook, November 2, to open a drive for funds for a medical clinic.

* * *

Dr. A. E. Hiebert and Dr. H. W. Brooks, Wichita, were speakers at the September meeting of the Sedgwick County Medical Assistants' Society.

* * *

Dr. Frederic B. Emery, Concordia, recently became a diplomate of the American Board of Surgery.

* * *

Dr. E. J. Ryan, Emporia, addressed the Lyon County Practical Nurses Association recently on the subject of "Glands."

* * *

Dr. Paul Brenneman, Hesston, left October 3 for Puerto Rico to spend two years at the La Plata Mennonite Hospital under the direction of the Mennonite Relief Committee.

* * *

Dr. Harold Bullock, Independence, discussed a proposal to build a tuberculosis sanatorium in southeast Kansas before the Lions Club in Independence recently.

* * *

Dr. H. L. Galloway recently began his 41st year in the practice of medicine in Anthony.

* * *

Among speakers at the annual one-day program of the Kansas Chapter of the American College of Surgeons, held at Hays in September, were Dr. Paul W. Schafer of the University of Kansas Medical Center, Dr. Irene Koeneke of Halstead, Dr. J. D. Colt of Manhattan and Dr. W. E. Grove of Newton. Dr. Colt was named president of the society and Dr. Orville R. Clark, Topeka, was elected secretary.

* * *

Dr. Charles R. Svoboda, Chapman, gave an instruction on first aid to bus drivers for the Dickinson County Community High School recently.

* * *

Kansans elected to office in the Kansas University Medical Alumni Association, at a meeting held in Kansas City last month, are: Dr. Robert Maxwell, Wichita, and Dr. Charles Underwood, Emporia, vice presidents; Dr. Mervin Rumold, secretary-treasurer; Dr. William Algie, chairman of the nominating committee. Dr. Franklin D. Murphy was speaker at the meeting.

* * *

A paper by Dr. John K. Fulton, Wichita, "Bronchial Asthma from a Therapeutic Point of View," published in July in the Journal of the Kansas Medical Society, was abstracted in the October issue of the Digest of Ophthalmology and Otolaryngology.

* * *

Dr. Howard E. Snyder, Winfield, was guest speaker at a meeting of the Sedgwick County unit of the American Cancer Society held in Wichita last month.

* * *

Dr. C. J. Kurth, Wichita, lectured on psychiatric therapy at the October meeting of the Sedgwick County chapter of licensed practical nurses.

* * *

Dr. Edward H. Hashinger, acting dean of the University of Kansas School of Medicine, led the discussion on postgraduate education at a meeting of the Association of American Medical Colleges at French Lick Springs, Indiana, October 29.

* * *

Dr. Ralph B. Earp, El Dorado, has announced plans to retire after having practiced for 53 years.

* * *

Dr. LeRoy A. Calkins, chairman of the Department of Obstetrics and Gynecology at the University of Kansas Medical Center, was installed as president of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons at a meeting held recently in Hot Springs, Virginia.

* * *

Dr. Donald R. Davis has discontinued practice in Olathe to begin a three-year residency in surgery at the Mayo Clinic, Rochester, Minnesota.

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(The Council on Pharmacy and Chemistry of the American Medical Association has adopted the following statement of Actions and Uses and of Dosage for publication in connection with a description of Banthine Bromide for inclusion in New and Nonofficial Remedies)

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Dosage.—Methantheline bromide is administered orally or parenterally by either the intramuscular or intravenous route. Parenteral administration is not advised for patients able to take the drug orally. The average initial adult dose, oral or parenteral, is 50 mg. For patients with considerable intolerance, 25 mg. may be employed. In the management of peptic ulcer, a beginning schedule of 50 mg. three times daily before meals and 100 to 150 mg. on retiring is suggested. However, the usual effective dose is 100 mg. four times daily, although some patients may require more or less than this amount. The dosage may be increased to tolerance, using dryness of the mouth as a guide, and adjusted to meet the individual response of patients. Maintenance dosage in peptic ulcer is usually considered to be about one-half the therapeutic level. In the management of other hypermotile or hypersecretory states, the dosage should be adjusted to the smallest amount which will relieve the symptoms. When spastic conditions are secondary to inflammatory or other organic lesions, therapy directed toward the cause should be employed whenever possible.

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Dr. Stanley Friesen and Dr. Mahlon H. Delp, members of the faculty at the University of Kansas Medical Center, were among the speakers at a meeting of the Missouri Academy of General Practice held in October.

COUNTY SOCIETIES

A meeting of the Wilson County Society was held at the Coffee Shop, Fredonia, on October 18. Guests for the evening were Mrs. Margaret Foster, Topeka, and Mrs. Linda Gowan, Caney, representatives of Blue Cross-Blue Shield, who announced plans for a community enrollment campaign in November. The society voted to assist in the campaign.

* * *

The Wyandotte County Society met at the city-county health center in Kansas City, October 16. The program was presented by two guest speakers. Dr. C. J. Brown, Jr., showed colored pictures to illustrate oral-facial trauma, and Dr. Harry Statland spoke on "Endocrinology—Facts and Fallacies."

* * *

Dr. and Mrs. C. W. Wilson, St. Francis, entertained 38 members of the Northwest Kansas Medical Society and Auxiliary at St. Francis on October 14. The physicians held an afternoon meeting at Dr. Wilson's office, and the speaker, Dr. S. E. Blandford, on the staff of the University of Colorado Medical School, discussed "Injuries and Infections of the Hands." Four new members were added, Dr. K. L. Knuth of Atwood, Dr. Glenn Martin of Goodland, Dr. B. Dan Ferguson of Bird City and Dr. Johnson of Norton.

* * *

Members of medical societies in the first councilor district held a joint meeting at Hiawatha, October 16, with 65 physicians and their wives attending. Dr. W. L. Anderson, Atchison, councilor of the district, presided. Dr. C. H. Benage of Pittsburg, president of the state society, spoke on "The Year's Objectives for the Medical Society," and Dr. Lucien R. Pyle, Topeka, discussed the work of the Volunteer Advisory Committee.

* * *

The Wilson County Society and its Auxiliary held a joint dinner meeting at the Emerald Room, Neodesha, on September 19. A business session was held later at the home of Dr. J. W. McGuire, and Dr. Glen McCray, Neodesha, was accepted as a new member.

* * *

Members of the Shawnee County Society met with dentists, pharmacists and representatives of pharmaceutical houses at White Lakes Country Club, Topeka, October 1. An informal dinner meeting was held.

* * *

A quarterly meeting of the Central Kansas Medical Society was held recently at the Ellsworth Country Club. Two guest speakers from Kansas City presented the program. Dr. Clarence Francisco spoke on "Common Orthopedic Problems," and Dr. George Herrman discussed "Rh Factor in Erythroblastosis and Exchange Transfusions."

* * *

Dr. Gerald B. Pees, Iola, was elected president of the Southeast Kansas Medical Society at a meeting held recently in Pittsburg. Dr. Eugene Myers, Iola, was named secretary.

* * *

A meeting of the Sedgwick County Society was held at the Allis Hotel, Wichita, October 9. Speaker of the evening was Dr. H. R. McCarrol, St. Louis, was discussed "Office Orthopedic Practice."

The 25th annual golf and skeet tournament of the society was held September 21 at Rolling Hills Country Club and the Wichita Gun Club. Dr. E. S. Edgerton won the president's trophy for golf, and Dr. F. F. Nyberg won the trophy for marksmanship.

* * *

A film on gastrointestinal cancer was shown at a meeting of the Butler County Society held in El Dorado, October 8. The society presented a gold key to Dr. F. F. Lemon, Douglass, who has completed 50 years in the practice of medicine, the third member of the group to achieve that record.

* * *

The Cowley County Society met October 18 at the Arkansas City Hospital. Dr. H. O. Anderson, Wichita, discussed two subjects, "Common Foot Troubles" and "Rehabilitation of the Hands."

New Faculty Members Appointed

Seven appointments to the faculty at the University of Kansas Medical Center were announced recently.

Dr. Leland D. Stoddard, formerly on the staff of Duke University School of Medicine, was named assistant professor of pathology and oncology. Appointed as instructors were Dr. Mary C. Colglazier, medicine; Dr. Nicholas Hotton, III, anatomy; Dr. Irene Cutter Keeling, pediatrics; Dr. William L. Mundy, medicine; Dr. John R. Patterson, pediatrics. Dr. Irving Kaas was named assistant instructor in medicine.

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Senior Thesis from the University of Kansas Medical School*

Acute Pulmonary Edema

A. Dean Burnett**
Kansas City, Kansas

DEFINITION: Pulmonary edema, a form of edema affecting the lungs, consists of an escape of fluid through the walls of the lung vascular tree into the interstitial tissue, the alveoli, and the bronchial tree.

Acute pulmonary edema is marked by rapid onset, rapid flooding of the lung alveoli with a serous or serosanguinous fluid, copious expectoration, and imminent asphyxia. To quote Henneman,[1] "Its nonspecific name has led to confusion with cardiac asthma, paroxysmal dyspnea or paroxysmal nocturnal dyspnea and chronic pulmonary edema. Cardiac asthma is constantly associated with heart disease; ancillary signs of congestive failure and asthmatic wheezes throughout the chest are usually present. Paroxysmal dyspnea . . . usually appears at night and seems to be the same as cardiac asthma except for the associated bronchial constriction. Chronic pulmonary edema is found in congestive heart failure and the condition is rarely characterized by the explosive onset often found in acute pulmonary edema."

A clinical description of acute pulmonary edema by Lewis[2] is as follows: "The onset . . . occurring usually at night and waking the patient from sleep; or it may come in the hours of daylight and wakefulness . . . In frank pulmonary edema, while breathlessness is usually urgent from the start, cough is more prominent, and frothy blood-stained fluid is freely or profusely expectorated. The march of events is often speedy. Cyanosis deepens rapidly; breathing becomes progressively more urgent and less effective. The attack may last at its height for hours. In fatal attacks the pulse becomes steadily weaker until imperceptible, the veins swell, and breathing becomes more gasping, less frequent, and then weaker until it ends.

"In fulminating form, the edema occurs so rapidly and is so intense that, within a minute or two of crying out in fear, the patient is drowned by the copious blood-stained fluid that pours into the respiratory passages and overflows frothing from the mouth and nose."

INCIDENCE: There is no satisfactory report on the incidence. However, Farber[3] states that acute pulmonary edema is one of the frequent causes of death found at autopsy in infants and children.

PATHOLOGY: The lungs at autopsy are heavy, water-logged, and pit on pressure. When incised, large amounts of clear or bloody-colored fluid exude from the cut surface. Histologically, the capillaries are dilated, and most of the alveoli are filled with an almost homogenous pinkish material. A few large hemosiderin-containing mononuclear cells may be present. The edema fluid contains protein, the concentration of which may be two to three per cent.

GENERAL CONSIDERATION OF FACTORS AS REGARDS ETIOLOGY AND PATHOGENESIS: The multiplicity of the causes which give rise to attacks of lung edema and the theories attempting to explain it are evidence that no one explanation offers an adequate mechanism accounting for all acute pulmonary edema attacks.

Structural arrangements in the lungs are significant in the development of pulmonary edema. (Portions of the following paragraphs relating to this aspect have been taken from Drinker[4, 5] and are presented herein with revision.)

In visualizing the capillary mesh of the lung, one should consider it overlaid on both sides by a delicate layer of extremely thin alveolar epithelium. It is the meat of a vital sandwich of which the enclosing outer layers are so thin as to be inconsequential in restraining diffusion of gas or of fluid from the capillaries. The vascular area provided by this net is maximally great, and the fall in pressure between pulmonary arterioles and capillaries is in propor-

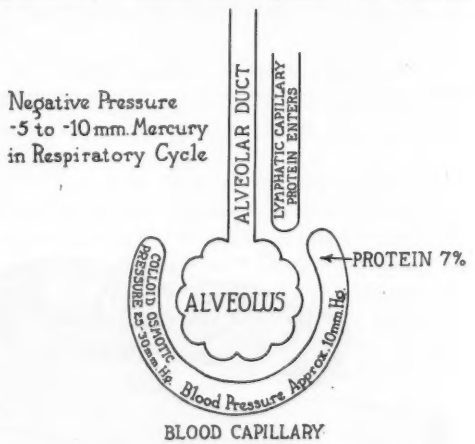

Fig. 1 - Schematic Representation of the Lung Structure

*This is one of 11 senior theses selected for publication by the Editorial Board from a group of 15 judged the best by the faculty of the University of Kansas School of Medicine.

**Senior Student, University of Kansas School of Medicine.

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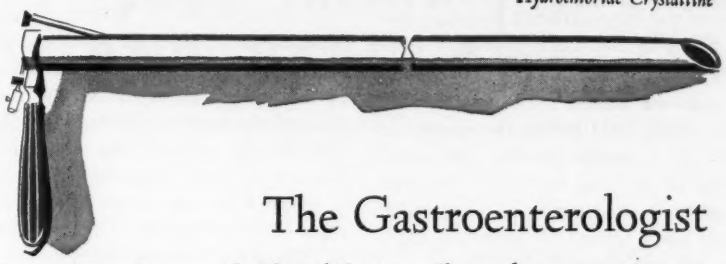

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tion. Arteriolar constriction might alter the distribution of lung blood to a degree, but in such a net as surrounds the alveoli, capillary reactions are minimal in regulating blood flow.

An important feature of the diagram in Figure 1 concerns the lymphatic system. Wherever lymphatics are found in the body, their principal function is the carrying off of small amounts of protein that have left the blood capillaries. Lymph vessels are distributed over the surfaces of the arteries, veins, and bronchi. However, their ends do not reach into the walls of the alveoli or surround alveoli as commonly thought, but stop as blind terminals in the walls of the alveoli ducts. Thus the ultimate saccular radicles of the lymphatic tree terminate just before the beginning of the largest portion of functional respiratory lung tissue.

This means that the alveoli septa must become turgescent with edema fluid before the lymphatics can be entered. So it seems possible that excess fluid filtered out of the lung capillaries can enter the alveoli air space about as readily as it can spread inside the alveoli tissue toward the lymphatic capillaries located at the beginning of the atria.

Furthermore, the arrangement of lymph channels for delivering their contents into the blood is curiously inadequate. All the lymph from the lungs, except a very small amount from the upper part of the lung, empties into the right subclavian vein by way of the right lymphatic duct, which is usually a short tube of not more than one millimeter inside diameter. This vessel is an astonishingly small bottleneck at the delivery end of a reservoir for accumulating fluid.

Mechanical factors play an important role in the dynamics of the lungs. The accompanying figure shows a capillary containing blood with a normal plasma protein content of approximately seven per cent. This protein exerts an osmotic pressure—the colloid osmotic pressure of the plasma of 25 to 30 mm. of mercury. In most systemic capillaries this force, which attracts water into the vessels, is opposed by the capillary blood pressure of about the same magnitude derived from the left ventricle. Consequently, a balance of forces controls movements of water and dissolved salts through the capillary endothelium.

In the lungs, the colloid osmotic pressure attracting water is necessarily 25 to 30 mm., but opposing capillary blood pressure is lower, 10 mm. being a fair value. However, the negative intrathoracic pressure exerts suction upon the walls of the blood capillaries. This pressure is —10 mm. in normal inspiration, and during heavy breathing may be —70 mm. A forcible expiration readily overcomes the negative intrathoracic pressure normally remaining at full expiration and may give rise to a positive pressure of 80 mm. Also at each normal inspiration, pressure in the trachea becomes negative (—3 mm.) and this effect extends through the bronchi into the alveoli. Obviously, under normal breathing there is an element of suction conducive to filtration in terms of 5 to 10 mm. This wipes out much of the balance for retention and absorption of water inherent in a system in which there is capillary pressure of 10 mm. opposing colloid osmotic pressure of 25-30 mm. With lung tissue and its aeration wholly normal, mechanical conditions producing significant capillary leakage do not occur.

In 1878 Dr. W. H. Welch conducted a series of experiments in which he produced pulmonary edema by compression of the left ventricle or auricle and by the thoracic aorta.[6] From this came the cornerstone of the mechanical theory of pulmonary edema. This theory assumes that through a heightened pressure of blood within the pulmonary circuit, fluid is squeezed into the tissues of the lung and out into the alveoli and bronchi. Other workers were unable to substantiate Welch's findings.

There have been numerous experiments concerned with the production of pulmonary edema by ligature of all or part of the pulmonary veins, by selective damage to the right ventricle or left auricle or ventricle, by ligature of the aorta below the diaphragm, and by ligature of the arch of the aorta with and without opening the thorax. These experiments indicate that pulmonary edema is *not* due simply to pulmonary hypertension.

Several investigators have proved that the severest grades of pulmonary edema might exist clinically and be produced experimentally without any evidence of increased pressure in the pulmonary circuit. Luisada[7] and Henneman[1] both refer to an editorial appearing in the British Medical Journal in 1941 which stated that as experimental and clinical observations on pulmonary edema had accumulated, it had become clear that the validity of the "back-pressure" or "left ventricular failure" theory of acute pulmonary edema could no longer be considered as established. Paine and Smith, writing in 1949, claim that evidence at hand provides no certain clue as to the mechanism of acute pulmonary edema from failure of the myocardium, and that pulmonary congestion alone does not appear to be the precipitating cause.[8]

However, Turnoff and Schnabel, writing in the 1951 edition of the *Cyclopedia of Medicine, Surgery Specialties,* state, "Diminished cardiac output and renal retention of sodium and water, occurring in patients with failure of the left-sided chambers, is

50 and Four

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the basis for pulmonary congestion. Sudden intensification of the engorgement leads to paroxysmal dyspnea, cardiac asthma, or acute pulmonary edema. Although not exclusively nocturnal in its appearance, acute edema at times develops as a transition from paroxysmal dyspnea or cardiac asthma by the production of massive lung edema and the rapid exudation of copious quantities of plasma into the interstitial tissue, the alveoli and the bronchial tree."[9]

Posture must be considered among mechanical factors which may be involved in the production of acute pulmonary edema. Perera and Berliner undertook a study "to see whether shift of body fluid to the blood stream, following rest in the horizontal position, could be correlated with the time of onset of paroxysmal dyspnea and might therefore be considered a factor in inducing acute left-sided heart failure." In their discussion of the study, they point out that "serum protein concentrations are considerably altered in health and disease by changes in position and muscular activity, that the decrease in serum protein concentration appears to be the result of hemo-dilution due to an increase in plasma volume, and that the close correlation between nocturnal hemo-dilution and attacks of paroxysmal dyspnea suggests that an increase in plasma volume is an important factor in the production of acute left-sided failure in individuals with organic disease."[10]

Drug, chemical, and inhalant factors should be considered. Acute pulmonary edema caused by adrenalin in animals shows notable similarities to that in humans. It occurs suddenly, is characterized by dyspnea, hypertension, tachycardia, and it soon causes the same rose-colored froth at the mouth and nose of the animals and similar alterations of the lungs. Henneman summarizes a systematic study done by Luisada on rabbits to determine the dosage of adrenalin necessary to kill the animals with acute pulmonary edema. Using such doses and even giving 50 per cent more adrenalin, many animals could be saved by a complex mixture of certain drugs. These drugs probably depress central nervous system centers. In adrenalin-produced pulmonary edema, the significant factor is supposedly the effect of hypertension—not a general or a pulmonary hypertension but rather aortic hypertension to a small degree and carotid and cephalic vessel hypertension to a larger degree. This hypertension activates a reflex arc that determines the sudden increase in the permeability of the pulmonary vessels and the consequent pulmonary edema.[1]

Johnson describes several experiments on rabbits with the chest open in which acute edema of the lungs did not occur following injection of adrenalin in doses that would normally produce edema were the chest closed. Presumably, alteration of the difference in pressure between external air and the air in the lungs was significant. He states that because of peripheral vaso-constriction and subsequent rise in systemic blood pressure, there results an obstruction to the circulation of the blood on the left side of the heart, but not on the right. This eventually leads to an over-distention of the capillaries of the lungs with blood. If the capillaries distend widely enough, the blood fluids escape through the capillary walls into the alveoli of the lung and edema is thus established.[11]

Paine and Smith point out that pulmonary edema following exposure to gaseous and parenteral toxins has raised the possibility of a toxic mechanism in the pathogenesis of lung edema. Newton concludes in experiments on cat heart-lung preparations that the pulmonary edema which occurred was primarily due to a toxic activity of the blood which was used. He states that in the pulmonary circulation it causes a rise in arterial pressure; the significance of this may be only as an index to a simultaneous toxic action upon the capillaries.[12] Other workers have suggested that the lung edema frequently occurring in shock is due to the action of an "H substance" carried to the lungs from the site of injury.

Experimental pulmonary edema has occurred following ingestion of potassium iodide, iodoform, histamine, and pilocarpine; by intravenous injections of chlorine water, prussic acid, amyl nitrite, and methyl salicylate; and after administration of thiourea and derivatives thereof.

Certain of the industrial gases and other substances that may cause pulmonary edema are: oxides of nitrogen, phosphorus oxychloride, phosphorus pentachloride, phosphorus trichloride, methyl bromide, chlorine, cadmium, and dust from specific alkaloids. Non-industrial toxins have been blamed in cases of alcoholism, from the effects of venoms and muscarine, and after administration of barbiturates, iodine, morphine, and prostigmine.

Anoxia is a recognized factor. Drinker states "It is generally acknowledged that two factors are fundamental in causing abnormal transudation in the lungs and pleural sacs. They are, first, sustained increase in pulmonary capillary pressure and, second, anoxia." He also remarks that when a tissue becomes anoxic, the capillary bed becomes larger and that often dilatation is accompanied by increased permeability, so that edema occurs. Increased pressure in the pulmonary capillaries does not readily cause recognizable pulmonary edema *unless* coupled with heightened permeability, most frequently due to anoxia.

A single, deep, subcutaneous injection of 50,000 to 90,000 U.S.P. units (approximately 300 to 500 mg.) of Depo* Heparin will give a lengthened coagulation time [illegible] times normal for about 24 hours.[1]

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1. Snyder, William T.: Long-Acting Heparin Preparations: A Useful Adjunct in Anticoagulant Therapy. U.S. Armed Forces Med. J. Vol. II, No. 1, Oct. 1951.

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Drinker further states that despite evidence purporting to show it, there is no reason to believe that the capillaries possess active powers of contraction and dilatation as is the case with many of the systemic vessels. This does not mean that their diameter does not change, nor that at times there are capillaries that are not conducting blood that may later be filled by a moving current. "These changes are, I believe, passive and depend on variations in the output of the right ventricle or a rise in pressure in the pulmonary veins." The lungs must meet the needs of the body when the cardiac output is only four liters a minute and also when it rises abruptly even to 30 liters.

The lungs, being larger than necessary to meet the resting needs of the body, often allow functional disruption of much lung tissue to proceed for a long time with quiet persistence, but without apparent disablement—a fact which too often leads to unhappy eventuality.

Think of one of the commonest clinical experiences—a patient safely in bed with a moderate degree of cardiac decompensation. There is little cause for alarm, a trifling dyspnea, slight cyanosis, and moderate edema of the lower part of the body. Yet, with respect to the lungs, this patient is getting oxygen through only a part of his lung tissue—a part which remains normal while other large sections filling with fluid cease to function. Even with removal of a lobe or an entire lung, the patient manages to carry on his respiratory functions well enough to live a reasonable life. He cannot shift suddenly into violent muscular effort, but he can be a relatively normal person if his physical activities are kept within bounds. However, what Meltzer called his "margin of safety" is gone. This is a fundamental conception for clinicians—the conception of the existence of protective mechanisms so beautifully ordered that doctors rarely realize how often they gamble upon safety margins.[5]

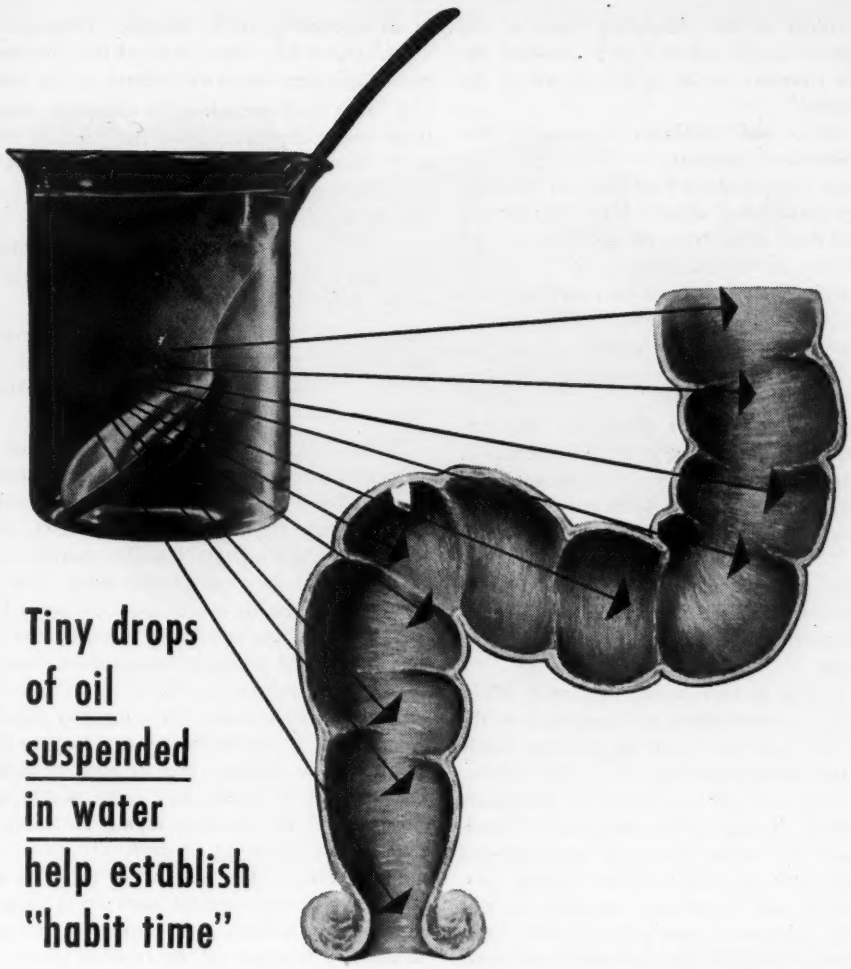
That the lung capillaries do not dilate adaptively as a result of the oxygen lack does not exclude them from the effect of anoxia—namely, increased permeability—and anoxia does make these vessels leak abnormally. It is an outstanding feature of oxygen lack that where it occurs "anoxia begets anoxia" and such is the striking case in the lungs.

Neurogenic factors play a role. Daly cites experiments demonstrating a rise in pulmonary arterial pressure following stimulation of the peripheral ends of the cut thoracic nerves from D2 to D7.[13] He refers to "the existence of functionally active pulmonary vasomotor nerves" and makes the general statement that "there is evidence of the existence of functionally active pulmonary vasoconstrictor fibers which appear to be adrenergic in character and also of pulmonary cholineric vasodilator fibers." It would seem that the action of the pulmonary vasoconstrictor fibers is more easily demonstrated than that of the vasodilator fibers.[14]

Farber reports that bilateral cervical vagotomy in rabbits soon leads to death, usually within 8 to 24 hours, from pulmonary edema. He concludes such changes are brought about by a combination of factors secondary to bilateral vagotomy among which are loss of the vagal innervation of the lungs and possibly laryngeal paralysis. He states that "loss of the power of pulmonary vasoconstriction deprives the pulmonary vessels of a mechanism necessary in the adjustment of pulmonary vascular dynamics." He also remarks that it appears that both the sympathetic and the vagal systems exert a control over the pulmonary and bronchial vessels. Experimental evidence suggests that neuropathic pulmonary edema in man is caused by disturbances, either central or peripheral, to the vasomotor control of the pulmonary vessels, resulting in one type of acute pulmonary edema on a neurogenic basis.[14]

Henneman cites the experiments of several workers who have produced pulmonary edema in animals by stimulation, injury, or extirpation of various nerves of the central and autonomic nervous systems. He states "merely mechanical and humoral factors on the heart and lungs cannot cause experimental pulmonary edema, which always occurs via a nervous mechanism, as in that produced by adrenalin and by ligation of the aorta. It is possible to cause pulmonary edema by modifying only the circulation of the head, and this is determined by a hypertension in the cephalic vessels. It is possible to cause pulmonary edema by electric stimulation of the nervous pathways."

Luisada[15] presents the neurogenic theory as follows: "According to this concept the attack is the result of acute congestion of the pulmonary vessels followed by transudation in the alveoli. The immediate cause of this condition is an intense stimulation of the vasodilator nerves of the lungs due either to a lesion of the nerve centers or to reflexes arising in the various viscera. The pulmonary edema in persons with skull injuries, with subarachnoidal hemorrhage, and with poliomyelitis is included in the first class, as the lesion is central. The pulmonary edema of drowning people, and, to a partial extent, that of soldiers subjected to war gases, should be included in the second class, as reflexes arising in the mucosa of the larynx, of the trachea, and of the bronchi seem to be the main causes of the attack. A multiple reflex probably occurs in hypertensive patients where the nerve receptors of the aortic arch, the carotid sinus, and of the heart wall are excited by distention. A simpler reflex causing typical at-



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tacks may occur in the stimulation either of the heart receptors (acute ischemia as in coronary occlusion) or receptors of the esophagus and of abdominal organs."

Other factors and conditions relating to pulmonary edema are of interest.

Drowning. Luisada states fluid found in the lungs of drowned persons and animals has a high protein content and must result from escape of serum into the alveoli, i. e., pulmonary edema.

Paracentesis. Edema has been described following thoracic or abdominal paracentesis. In 1943 Vidal et al reported contralateral pulmonary edema was provoked by creation of an extrapleural pneumothorax.

Uremia. Acute pulmonary edema has been observed frequently during the course of uremia. It rarely occurs, however, in pure epithelial nephritis. One worker believes that the pulmonary edema of renal disease is due to accompanying cardiac weakness.

Pregnancy and toxemia of pregnancy. Certain authors have stressed the importance of the relationship between pregnancy and pulmonary edema in some cases.

Central nervous system damage or disease. Weisman and Master report edema and congestion of the lungs have developed to a severe degree immediately after an intracranial hemorrhage.[16, 17] The following have been cited also as causes of pulmonary edema: insulin hypoglycemic coma, brain tumor, head wounds and trauma, diseases of the peripheral vagus nerve, acute ascending myelitis, tetanus, cerebral embolism and thrombosis, vertebral injuries, encephalitis, polyneuritis and polyradiculitis, tabes dorsalis, lumbar puncture, phrenic-nerve crush under local anesthesia, cervical sympathectomy, laminectomy, emotional disorders, and hysteria.[1, 18, 19]

Infections. The following are regarded as having initiated pulmonary edema: acute rhuematic fever, puerperal fever, typhoid fever, measles, scarlet fever, and whooping cough.

Cardiovascular diseases. Cardiovascular diseases in which pulmonary edema may occur are: aortic regurgitation, aortitis, angina pectoris, coronary thrombosis, pericarditis, mitral stenosis, cardiac failure, hypertension, infarction, wounds of the heart, and cardiac aneurysm.

Miscellaneous. Pulmonary edema has been reported in association with or initiated by: coitus, shocking news, allergy, thyroid storm, epileptic seizures, beriberi, gout, reflexes from internal organs during operations, distention of abdominal organs, shock, intravenous injections of muscarine, iodine, bile salts, burns, intestinal obstruction, and manipulation of stellate ganglia. Burford and Burbank,[20] in an interesting article entitled "Traumatic Wet Lung," report they have observed two fundamental physiologic phenomena with regard to chest wounds:

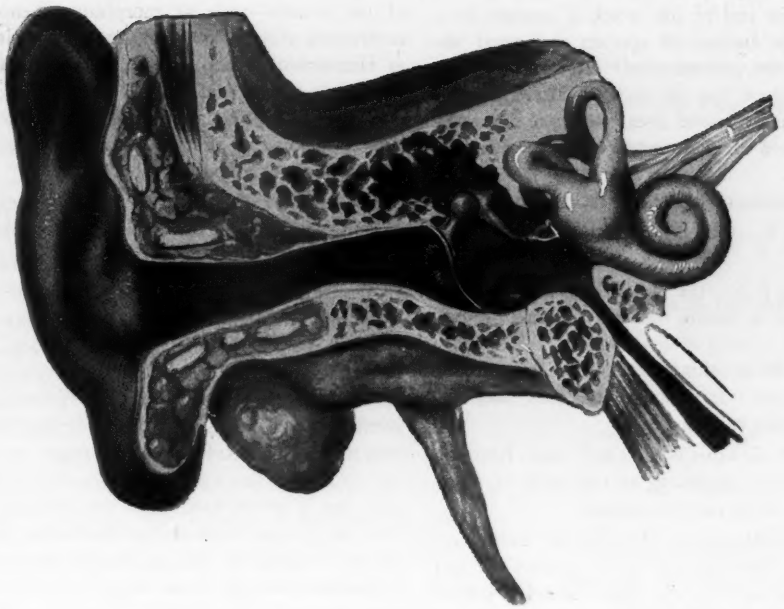
1. That in all wounds of the chest to a greater or lesser degree depending upon the type and severity of the lesion, the lung tissue reacts to produce more than its normal amount of interstitial and intra-alveolar fluid.

2. That in all wounds of the chest the bronchopulmonary tree not only has more fluid to rid itself of, but becomes less capable of doing so.

They state that the result of these two phenomena may be the factor upon which the outcome of the patient's life will depend. (For their recommended treatment, see under THERAPY.)

SYMPTOMS AND SIGNS: An attack of acute pulmonary edema may occur at any time. Restlessness, weakness, dry non-productive cough, and precordial oppression may precede the attack. If this occurs at night, a nightmare usually precedes it. The onset may awaken the patient from sleep with breathlessness usually urgent from the start. Cough becomes prominent and frothy blood-stained foamy fluid is freely or profusely expectorated. This fluid may attain a volume of 500 cc. or even 1000 cc. within 30 to 60 minutes. Vomiting may occur. Frequently there is profuse sweating, paroxysms of suffocation with cyanosis, and urinary incontinence. The sequence of events may occur within a very short interval, with cyanosis rapidly becoming more marked and breathing progressively more urgent and less effective. In a fulminating form, the edema may be so intense and of such rapid onset that within a few minutes after crying out in fear, the patient "is drowned by the copious blood-stained fluid that pours into the respiratory passages and overflows frothing from the mouth and nose."

The physical findings characteristic of this condition are the presence of numerous coarse bubbling rales throughout the lungs and feeble heart sounds. The rales vary markedly, from the minute alevolar rales to the gurgling sounds caused by the foam in the trachea. The alveolar rales are frequently more abundant at the bases. The pinkish frothy blood-stained fluid which may pour from the mouth or be rapidly expectorated is practically pathognomonic. Except in attacks which are connected with myocardial infarction (usually) and mitral stenosis (sometimes), the pulse is full and rapid, and the blood pressure is normal or high, the latter frequently being higher than before the attack. The temperature remains normal during the attack but may be elevated following it (due to readsorption of proteins from the lungs). The blood NPN may be increased. This increase may last two to three



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days even in patients without apparent renal lesions.

The duration of an attack of pulmonary edema is variable, lasting from but a few minutes to several hours. The end of the attack is marked by a decrease in the amount of sputum and rapid improvement in the general condition of the patient. Rales in the chest may be detected afterwards for minutes to hours in some cases, and there is no correlation between their presence and the suffering of the patient.

Luisada[21] would classify the attacks as follows:

Abortive P. E.—the entire attack lasts only a few minutes.

Fulminating P. E.—the patient dies before a physician is called or during transportation to the hospital.

Prolonged P. E.—this type usually is connected with myocardial infarction. The attack may last from many hours to many days.

Unilateral P. E.—this is extremely rare. It usually is associated with pathology of one lung or a unilateral lesion of the nervous system.

ROENTGENOLOGIC FINDINGS: Nessa and Rigler[22] in discussing the x-ray picture of pulmonary edema refer to the classic butterfly-shaped, fairly symmetrical density of both lungs. This is greatest at the hilus and gradually fades out toward the periphery; the apexes and extreme bases may be clear. The density is homogenous and obliterates all structural detail in the lung. The fluid collects in groups of alveoli, resulting in an irregular stippled density on the roentgenogram. Often the edema is predominantly on one side or entirely unilateral. The condition is distinguished from pulmonary congestion by the presence of enlarged hilar shadows and dilated vessels throughout the lung fields. It is further distinguished from a massive atelectasis in which there is elevation of the diaphragm, shift of the mediastinum and collapse of the chest wall. Lateral films reveal the congestion and edema to be central and not peripheral, in contrast to the congestion in pneumonia, which is usually peripheral in onset. According to Burford and Burbank, x-ray findings may be completely absent even in the presence of marked auscultatory moisture.

THERAPY: Opinions vary as to proper therapy for this condition as would be expected considering the confusion that exists regarding the cause(s) of the condition.

Naturally, followers of the heart-failure theory advocate drugs stimulating the myocardium, vasomotor center, peripheral arteries, carotid sinus and respiratory center—such as digitalis, ouabain, strophanthin, camphor, Coramine, caffeine, metrazol, and ether.

Sedatives and narcotics are recommended by most clinicians to depress the central nervous system, the autonomic nervous system, and smooth-muscle fibers of the vessels—such as morphine, phenobarbital, chloretone, chloral, atropine, and papaverine. And as Henneman points out, the foregoing implies a belief in a neurogenic theory. Luisada suggests that in some ways the sedative drugs are antagonistic to the stimulants and that therefore, if the one group is used, the other should not be.

The action of the stimulating drugs is regarded as so complex that their use is objectionable even when the theory of left ventricular failure is accepted.

Among mechanical procedures are those designed to lessen pulmonary congestion or to improve oxygenation of blood. Therefore venesection, application of tourniquets, and positive pressure respiration may be employed. Drinker recommends the administration of oxygen. He states that the reasons why there is still too little use of oxygen are (1) failure to realize that anoxia is essentially progressive and that it is not used until the physician is sure that the patient needs it, by which time he has incurred a handicap that is usually insurmountable. "The time to begin to use oxygen is before there is any certainty it is needed." And (2) the present methods for administering oxygen are expensive and inefficient. He recognizes that there is no drug that gives such great subjective relief and valuable rest in dyspnea as does morphine, but points out that this drug dampens the respiratory center and thus promotes anoxia. He therefore advocates the use of oxygen with morphine to allow all possible benefit from the sedative effect without the progression of anoxia.

He concludes that the two principles that the laboratory has learned are the master words in oxygen therapy: anoxia begets anoxia, and if real benefit is to be expected from oxygen, it should be used before the need is certain.

Harrison[23] states that when seizures are relatively mild, no treatment is required other than assumption of the upright position and administration of codeine to control cough. (The presence of wheezing indicating cardiac asthma is usually an indication for aminophylline, which may lessen dyspnea even when the obstructive component is not obvious. The usual dose is 0.5 gm. (7½ gr.) administered either intravenously over a five-minute period or by rectum when dyspnea is mild. When the rectal route is employed, sodium theophylline glycinate ("Glytheonate") appears to be more effective and better tolerated, the usual dose being 0.8 gm. (12 gr.) as a suppository.

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are: morphine, especially in patients with pain; phenobarbital in cases with central nervous system lesions and no abnormality of blood pressure; and papaverine in patients with coronary occlusion. Luisada concludes that chloretone, chloral, sodium phenobarbital, papaverine hydrochloride, atropine sulfate, and morphine sulfate may safely be injected intravenously, and that during the acute attack digitalization and stimulants should be avoided. He states that "intravenous injection of either chloral hydrate or phenobarbital in combination with morphine gives the best result. Atropine should be avoided unless bradycardia is present."

His therapy for the prevention of attacks includes: digitalis for subacute insufficiency of the heart; morphine subcutaneously, in addition to atropine and phenobarbital, following skull trauma, since patients usually develop pulmonary edema six to twenty-four hours after injury; and opium and belladonna by mouth in cases of recurrent pulmonary edema. He also suggests chloral hydrate by mouth or by rectum, phenobarbital, and aminophylline as good prophylactic therapy.[21]

Luisada remarks that some of the drugs which are useful in certain types of pulmonary edema might be detrimental in others. Morphine, barbiturates and chloral are advocated in cardiac patients but are not advocated in pulmonary edema following injury to the central nervous system because of their depressing action on the nerve centers. Intravenous strophanthin might cause ectopic rhythms in pulmonary edema following coronary occlusion. Venesection, mercurial diuretics, spinal anesthesia, and possibly morphine should not be used in pulmonary edema accompanied by shock because they further reduce venous return and cardiac output. Oxygen under pressure may not be well tolerated by patients with emphysema.

According to Burford and Burbank, the time-honored method of controlling thoracic wall pain in traumatic injury to the chest wall has been adhesive strapping and the generous use of morphine which they claim is a physiologically unsound approach. They state while morphine, by raising the pain threshold makes the patient less aware of pain, it actually diminishes the cough reflex and does not aid in the establishment of a painless cough mechanism. When adhesive strapping is used, the existing anoxia is further increased by limitation of the motion of the chest wall. In addition, it is an actual handicap to the propulsive mechanism of cough. Accordingly, the therapy which they propose is abolition of pain and physiologic remobilization of the chest wall. Temporary intercostal nerve block and thoracic paravertebral sympathetic nerve block are suggested. In some patients so flooded with bronchopulmonary secretion and so exhausted by their trauma, tracheobronchial catheter aspiration may be employed.

The use of antifoaming agents in the therapy of paroxysmal pulmonary edema is of interest. Luisada states it has been known for a long time that fairly large amounts of fluid may be tolerated in the respiratory passages as long as no foam is formed. When the latter develops, increased volume of the air-fluid mixture and modified physical properties lead to severe effects by blocking the small bronchi. The stability of a foam is based upon the character of the air-fluid interface, namely on the surface tension of the fluid, and any substance capable of modifying the surface tension in such a way as to decrease the foam, is called an anti-foaming agent. In experiments performed on rabbits in which a series of anti-foaming agents were administered by inhalation in order to decrease the severity of pulmonary edema caused by a standard dose of intravenous adrenalin, poorly volatile drugs (heavy alcohols, Span 85) failed to exert any favorable effect. However ethyl alcohol exerted an important favorable action, due to the anti-foaming property of alcohol; its action on the central nervous system, though slight because of the small dose used, may have enhanced the effect. The favorable effect of alcohol was comparable to that of morphine. Combination of morphine by injection with alcohol by inhalation gave excellent results, equivalent to those obtained by morphine plus oxygen under pressure. The inhalation of alcohol vapor in animals with pulmonary edema induced by other methods than by intravenous adrenalin gave striking results, entirely preventing the development of pulmonary edema in some instances. Clinical treatment with alcohol by inhalation is now being tried.[21, 24]

For the benefit of those who are faced with the problem of treating acute attacks of congestion and edema of the lungs, and dyspnea, the following paragraph is quoted from Harrison:[23] "In the case of a severe seizure, energetic therapy is life-saving. Morphine should be employed in large doses (15 to 30 mg.) in young and middle aged adults, but should be used with caution in the elderly, and in persons with impairment of hepatic function. In the younger subjects with very severe dyspnea, the intravenous route is to be employed. Venesection (about 500 ml.) should be carried out immediately and may need to be repeated in desperate cases. Venostasis (trapping blood in the legs by the applications of pressure cuffs to the thighs) is likewise useful. Oxygen is urgently indicated. Digitalis in the form of rapidly-acting Digitalis lanata ("Cedilanid," 0.8 to 1.2 mg.) or strophanthin (ouabain) should be administered intravenously to

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Summary

No one explanation offers an adequate mechanism accounting for all acute pulmonary edema attacks.

Increased pulmonary tension, lowered concentration of serum proteins (which may result in hemodilution from assumption of supine position), increased pulmonary capillary permeability, and anoxia are causative factors of the condition.

The termination of the lung lymph vessels in the walls of the alveolar ducts and not in the alveolar walls, and the questionable adequacy for drainage by the right lymphatic duct are significant factors.

Chemical substances, drugs, inhalants, trauma, and the stimulation of various nervous reflexes associated with the pulmonary vascular tissues play a role.

The symptoms and signs consist of sudden onset of dyspnea, cough, expectoration of frothy blood-stained fluid, the presence of numerous coarse bubbling rales throughout the lungs, and feeble heart sounds.

Roentgenologically, acute pulmonary edema is characterized by the butterfly-shaped, fairly symmetrical density of both lungs which is greatest at the hilus and gradually fades out toward the periphery.

The therapy consists of the use of morphine, oxygen, venesection, venostasis, and digitalis.

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BOOK REVIEWS

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The tablet material (2 gm. equivalent to 30.8 grains Doraxamin) was added to 150 cc. artificial gastric juice and stirred at 37 degrees C. Every 10 minutes there was removed 20 cc. of the mixture which was replaced by 20 cc. of fresh artificial gastric juice. At regular intervals the pH of the mixture was determined with a Beckman pH meter.

FOR THE TREATMENT of peptic ulcer and hyperacidity, the market has long afforded neutralizing agents which are satisfactory to a degree. Frequently, however, a dosage sufficient for prompt, lasting pain relief brings in its wake a discouraging acid rebound.

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Karl J. Waggener, M.D. **Wendell T. Wingett, M.D.**

faults, I feel, is the lack of clear, differential diagnosis. This book I highly recommend for individuals who are beginning their introductory phase into the broad field of psychiatry, for it serves as a good stepping-stone to further reading in psychiatric literature.—*E.E.B.*

* * *

The Changing Years. By Madeline Gray. Published by Doubleday and Company, New York. 224 pages. Price $2.75.

This is an honest effort to present in understandable form the bodily changes responsible for "menopausal symptoms." Too much time and detail are spent on certain phases of these changes, but on the whole the book should have definite value for the doctor in management of some of his patients complaining of "the change," since it can be used to supplement his verbal remarks.—*R.L.N.*

* * *

Review of Physiological Chemistry. Third Edition. By Harold A. Harper. Published by University Medical Publishers, Palo Alto, California. 260 pages, 13 figures. Price $3.50.

This book is an excellent, up-to-date, and concise outline of the subject of biochemistry, written principally as a supplement to standard texts. It is not sufficiently detailed for use as a text in medical biochemistry, but might conceivably be used for shorter courses in this subject. It might be used to advantage by the physician or basic medical scientist for review purposes or by interested non-medical scientists as an introduction to the subject. There is an emphasis on applications in the medical field but discussions of basic ideas are not lacking. Its usefulness could have been enhanced, perhaps, by the inclusion of specific references to the topics discussed. General references to current texts of general biochemistry and to books of special topics in the field are included in the back.

A number of well-chosen illustrations are used and the type and paper are satisfactory. It is well indexed and, hence, might serve as a handbook of the facts and principles of physiological chemistry. —*H.J.G.*

* * *

Clinical and Roentgenologic Evaluation of the Pelvis in Obstetrics. By Howard C. Moloy. Published by W. B. Saunders Company, Philadelphia. 119 pages, 68 figures. Price $2.50.

This monograph is a good general review of variations in the female pelvis which are of obstetrical significance. The author discusses the mechanism of labor, especially in regard to forceps operation, as related to different pelvic types. He presents his roentgenological method for attempting to predict cephalopelvic disproportion of the inlet and midpelvis. As yet, no satisfactory method for measurement of the fetal head in the breech presentation has been reported.—*C.A.H.*

ANNOUNCEMENTS

The U. S. Atomic Energy Commission will offer eight fellowships in industrial medicine for the 1952-1953 academic year, continuing a program begun two years ago. Awards are for one year's academic training at approved institutions. After completion of this training, fellows will be eligible to apply for a second year's in-plant training at one of the major installations of the A.E.C. The stipend for the first year will be $3,600, plus tuition and laboratory fees, and for the second year, $5,000.

Applications will be accepted up to January 1, 1952. The address is A.E.C. Fellowships in Industrial Medicine, University of Rochester, School of Medicine and Dentistry, Rochester, New York, Attention: Dr. H. A. Blair.

* * *

The American Urological Association offers an annual award of $1,000 (first prize of $500, second prize of $300 and third prize of $200) for essays on the result of some clinical or laboratory research in urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to men in training to become urologists. Closing date is February 15, 1952. Complete information may be secured from Dr. Charles H. de T. Shivers, Boardwalk National Arcade Building, Atlantic City, New Jersey.

* * *

The U. S. Navy announced recently that it would make 176 naval hospital internships available to medical school students graduating in 1952. Prospective interns must meet all requirements for a commission and must serve a minimum of 24 months of active duty from the date they start intern training. Application may be made at any time up to January 7, 1952.

Successful appointees will be commissioned lieutenants, junior grade, and will receive pay and allowances in that rank during internship. When ordered to other duty on completion of internship, they will qualify for an extra $100 per month. Other benefits include a uniform allowance of $250, transportation for dependents and household effects to duty stations, and opportunities for advanced professional training.

Complete information may be obtained from the Surgeon General of the Navy, Bureau of Medicine and Surgery, Navy Department, Washington 25, D. C.

Relationship of Stress to Autonomic Lability

Studies in psychosomatics have shown that functional disorders often are a result of the patient's inability to adjust to emotionally stressful situations (stressor factors).

Nervous tension and chronic anxiety, discharged through a labile Autonomic Nervous System, can cause somatic disturbance. [1,2] Such states may involve any one of the organ systems or several at one time. [1,3] The outline below is designed to relate gastrointestinal and cardiovascular symptomatology to the exaggerated response of the autonomic nervous system.

	Physiologic Effects of Autonomic Discharge	
	Sympathetic	Parasympathetic
Gastro-intestinal System	Hypomotility Intestinal Atony Hyposecretion Reduced salivation	Hypermotility Gastrointestinal spasm Hypersecretion
Cardio-vascular System	Rapid heart rate Peripheral vaso-constriction	Slow heart rate Vasodilatation
Functional Manifesta-tions	Palpitation Tachycardia Elevated blood pressure Dry mouth and throat	Heartburn Nausea-vomiting Low blood pressure Colonic spasm

The data here tabulated is from references 3,4,5,6,7, given below.

When the clinical picture is suggestive of functional disorder, the diagnosis is supported by the presence of the following indications of autonomic lability:

Variable Blood Pressure
Body Temperature Variations
Changing pulse rate
Deviations in B. M. R.
Exaggerated Cold Pressure Reflex
Oculo-Cardiac Reflex Abnormalities
Glucose Tolerance Alterations

Therapy in these cases is directed toward: 1) relieving the somatic disturbance to prepare the patient for psychotherapy*; 2) guidance in making adjustment to stressful situations and correction of unhealthy attitudes.

*Drug treatment using adrenergic and cholinergic blocking agents in conjunction with sedatives. [8,9,10]

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Anesthesiology. Sec., Dr. Curtiss B. Hickcox, 80 Seymour St., Hartford 15, Conn. Oral, Memphis.

Dermatology and Syphilology. Sec., Dr. George M. Lewis, 66 East 66th St., New York 21. Written, Chicago, March 1952. Oral, Chicago, May 1952. Final date for filing, Feb. 1, 1952.

Internal Medicine. Sec., Dr. William A. Werrell, 1 West Main Street, Madison 3.

Obstetrics and Gynecology. Sec., Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6. Oral, Chicago, June 7-13, 1952. Final date for filing, Feb. 1, 1952.

Ophthalmology. Sec. Dr. Ewin B. Dunphy, 56 Ivie Road, Cape Cottage, Maine. Written, Feb. 4-5, 1952, 25 centers. Oral, Oct. 8-15, Chicago.

Otolaryngology. Sec., Dr. Dean M. Lierle, University Hospital, Iowa City. Oral, Chicago, Oct. 8-12.

Pathology. Sec., Dr. Robert A. Moore, 1402 S. Grand Blvd., St. Louis.

Pediatrics. Sec., Dr. John McK. Mitchell, 6 Cushman Road, Rosemont, Pa.

Physical Medicine and Rehabilitation. Sec., Dr. Robert L. Bennett, 30 N. Michigan Ave., Chicago.

Plastic Surgery. Office of the Board, 4647 Pershing Ave., St. Louis 8.

Preventive Medicine and Public Health. Sec. Dr. Ernest L. Stebbins, 615 N. Wolfe St., Baltimore.

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Psychiatry and Neurology. Sec., Dr. David A. Boyd, Jr., 102-110 Second Ave., S.W., Rochester, Minn.

Radiology. Sec., Dr. B. R. Kirklin, 102-110 Second Ave., S.W., Rochester, Minn.

Surgery. Sec., Dr. J. Stewart Rodman, 225 South 15th St., Philadelphia. Written, various centers, March 1952. Final date for filing, Dec. 1, 1951.

Thoracic Surgery. Sec., Dr. William M. Tuttle, 1151 Taylor Ave., Detroit.

Urology. Sec., Dr. Harry Culver, 30 Westwood Road, Minneapolis 16. Chicago, Feb. 9-13, 1952.

The National Federation of Obstetric-Gynecologic Societies has reconstituted itself as the American Academy of Obstetrics and Gynecology. The academy was incorporated on August 4, 1951, and the constitution and by-laws were adopted at a meeting held at Hot Springs, Virginia, on September 5.

The first business meeting of the academy will be held at the time of the meeting of the American Congress of Obstetrics and Gynecology in Cincinnati, March 31-April 4, 1952. The first annual clinical meeting will be held in Chicago during the winter of 1952-1953.

Applications for fellowship may be obtained from the secretary's office, 116 South Michigan Avenue, Chicago 3, Illinois.

* * *

The American Society for the Study of Sterility announces the opening of the 1952 contest for the most outstanding contribution to the subject of infertility and sterility. A cash award of $1,000 will be given. Essays may be submitted until March 1, 1952. Complete information may be obtained from the society, 20 Magnolia Terrace, Springfield, Massachusetts.

* * *

The tenth annual meeting of the American Academy of Dermatology and Syphilology will be held in Chicago at the Palmer House, December 8-13. In addition to the regular sessions, there will be 36 informal discussion group meetings.

* * *

The American Trudeau Society, medical section of the National Tuberculosis Association, invites the submission of scientific and clinical titles for presentation at its annual meeting to be held at the Hotel Statler, Boston, the week of May 26, 1952. Abstracts limited to 300 words or completed papers will be reviewed by a committee for selection of the most interesting material. Subjects will include all aspects of tuberculosis, non-tuberculous respiratory and cardiac disease, and the results of investigative work with bearing upon these subjects. Material should be sent to the Chairman, Medical Sessions Committee, American Trudeau Society, 1790 Broadway, New York City.

* * *

The National Foundation for Infantile Paralysis announces the availability of a limited number of postdoctoral fellowships to candidates whose interests are research and teaching in medicine and the related biological and physical sciences. The fellowships cover a period of from one to five years. Stipends range from $3,600 to $7,000 a year. Complete information on qualifications and applications may be obtained from Division of Professional Education, National Foundation for Infantile Paralysis, 120 Broadway, New York 5, New York.

ABSTRACTS FROM CURRENT LITERATURE

Acute Small Bowel Obstruction

The Management of Acute Small Bowel Obstruction. By Earle B. Mahoney, Charles D. Sherman, Jr., N. Y. St. Jnl. Med., Feb. 1, 1951.

Only by early recognition of intestinal strangulation whether it is the simple mechanical obstruction (which may be prepared for elective surgery by intestinal intubation and replacement therapy), or the strangulated obstruction (which demands early operation), can the proper management of the patient be handled successfully. It is often difficult to differentiate between these two conditions, but there are certain diagnostic features which are helpful. In the presence of intestinal strangulation, the onset of pain is often abrupt and dramatic, severe and continuous. There are signs of shock and low blood pressure, and peritoneal irritation develops rapidly with tenderness, muscle spasm, and rebound pain. A rapidly rising white blood count is an excellent guide to a serious underlying picture.

The x-ray picture of the abdomen is usually one of the most helpful diagnostic aids in bowel obstruction. Here, when the dilated loops are filled with fluid and gas, shadows form the "ladder" pattern and outline the contour of the dilated bowel. Abdominal paracentesis carefully performed with a small gauge needle is a helpful and safe diagnostic procedure, also.

If diagnosis can be made with certainty that no strangulation exists, the initial phase of treatment is directed toward intestinal decompression and restoration of fluid and electrolyte balance.

The ideal method in intubation is to pass a tube eventually to the point of obstruction. The Miller-Abbott and the Harris mercury-weighted tube are most widely used. The writers prefer the Harris tube. Positioning and passing the tube under the fluoroscope has been one of the most successful ways in which to facilitate the passage of the tube. Some advantages gained by intubation are less trauma to the bowel, postoperative paralytic ileus is minimized, and the patient may be fed an adequate diet.

As soon as the diagnosis of intestinal obstruction has been made, blood should be obtained for chemical analysis. A large amount of chloride is lost by intestinal drainage, and should be replaced by Ringer's solution. Also, a great deal of protein is

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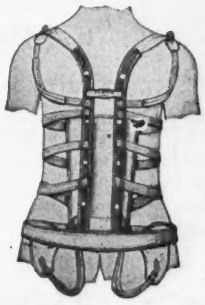

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lost in the dilated bowel, and should be replaced by plasma or whole blood. The loss of potassium in obstruction is probably of clinical significance only in the patient who has had prolonged drainage.

If non-strangulating obstruction is seen early and before marked distention has developed, early operation is preferred. Where there is marked distention, the patient is seen late, or there is no evidence of strangulation, or, if the patient has recurrent adhesive obstruction, then there should be prolonged preoperative preparation. If the site of the obstruction can be localized, transverse or oblique incisions are preferred to longitudinal because of the smaller incidence of postoperative wound disruption. If there is any doubt as to the diagnosis, or any signs of strangulation, then early operation is mandatory.

Often it is difficult to decide whether or not a strangulated segment should be resected. The writers prefer to perform a resection if there is any question about the viability of a segment. The manipulation of the distended bowel should be avoided as much as possible, and it is usually possible to pick up a collapsed loop and trace it to the point of obstruction. The Noble method of plication in recurrent obstruction is very useful. It would also seem indicated to give all patients chemotherapy, particularly those having closed loops and resections.—*J.J.H.*

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